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AN INVESTIGATION INTO THE DIAGNOSTIC VALUE OF PLEURAL FLUID SUGAR IN THE AFRICAN

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The low level of glucose in the cerebrospinal fluid of patients with tuberculous meningitis is frequently of assistance in differentiating tuberculous from other types of meningitis. It has been reported by two investigators[1,2] that, similarly, a low glucose-content of the pleural fluid (less than 60 mg. per 100 ml.) would indicate a diagnosis of tuberculous pleurisy.

Effusion into the pleural cavity is very common in the African and the present study was undertaken in order to determine whether glucose estimations in pleural fluids would be of value in differentiating between tuberculous and non-tuberculous pleurisy.

Subjects and Methods:

The investigation was carried out on 50 African patients, their ages varying from 19 to 80 years. The diagnosis was made by means of chest X-ray, sputum, urine and blood examinations.

In this series of 50 patients with pleural effusion, 27 were tuberculous. Of these diagnosed as pulmonary tuberculosis, the tubercle bacillus was found in the sputum or pleural fluid in 16. In the remaining 11, although the organism was not found, the clinical features, laboratory findings and therapeutic responses were such that there appeared to be no doubt of their tuberculous origin. In the 23 non-tuberculous patients the most important causes of pleural effusion were pneumonia, cardiac failure, carcinoma of the lung, and pulmonary infarction.

The fluid obtained from the pleural cavity, before treatment was instituted, was placed into 2 containers:

(*a*) A sterile container for bacteriological investigations including guinea pig inoculation, and total protein, specific gravity and chloride estimations, and

(*b*) a sugar tube containing a mixture of sodium fluoride and thymol to prevent clotting and glycolysis.

The true sugar content was determined by the method described by King and Garner.[3]

Results

The results of this investigation are shown in Table I. Repeated examinations gave similar results.

TABLE I. PLEURAL FLUID SUGAR RESULTS IN 50 AFRICANS

Glucose Levels mg./100 ml.	*Tuberculous*	*Non-tuberculous*
0–10	—	1
11–20	2 (2)	2
21–30	1 (1)	1
31–40	2 (1)	–
41–50	2 (2)	1
51–60	5 (1)	1
61–70	3 (1)	1
71–80	5 (3)	4
81–90	2 (2)	6
91–100	4 (2)	6
>100	1 (1)	–
Mean (mg./100 ml.)	67	71
Total No. of cases	27	23

Figures in parentheses indicate the number of patients in whom tubercle bacilli were isolated from sputum or pleural fluid. As can be seen from the table, the pleural fluid sugar was more than 60 mg./100 ml. in 15 of 27 tuberculous and less than 60 mg./100 ml. in 6 of 23 non-tuberculous cases. Statistical analysis of the results shows that there is no significant difference in the pleural fluid sugars in the tuberculous and non-tuberculous groups.

DISCUSSION

Calnan *et al.*[2] give a summary of recent investigations on the diagnostic value of the glucose content of pleural effusions including their own studies on 61 cases. They conclude that a glucose level of less than 60 mg./100 ml. is found only in tuberculous effusions. Our own findings

do not support this. Of the 23 non-tuberculous cases 6 had pleural fluid sugars of less than 60 mg./100 ml. The final diagnosis in these 6 cases was: Pulmonary embolus (sugar 10 mg./100 ml.); stab wound (sugar 17 mg./100 ml.); pneumonia (sugar 19 mg./100 ml.); pneumonitis (sugar 30 mg./100 ml.); carcinoma of the lung (sugar 34 mg./100 ml.); congestive cardiac failure + hypertension + cerebral haemorrhage (sugar 46 mg./100 ml.).

Gelenger and Wiggers[1] in their series of 31 cases, of which 21 were non-tuberculous, have also described a pleural-fluid sugar of 40 mg/100 ml. in a bronchogenic carcinoma.

In view of these findings, viz. that a sugar level of less than 60 mg./100 ml. can be found in non-tuberculous pleurisy, it would appear that no diagnostic value is to be gained from assessing the sugar content of the pleural fluid in the African.

SUMMARY

1. The glucose levels were determined on 50 Africans with pleural effusion with the object of determining whether the test would be useful in differentiating between tuberculous and non-tuberculous pleurisy.

2. From the results obtained it would appear that this estimation is of no diagnostic value.

I wish to thank the Director of the Institute for his interest and for facilities for carrying out this work, Dr. I. J. Grek for his help and encouragement, and the physicians of Baragwanath, Coronation, Crown Mines and Libanon Hospitals who kindly assisted in this investigation.

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THE MIDDLE CLASSES AND THE RISING COST OF LIVING*

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We all know that statistics can be made to prove almost anything. And statistics relating to wages, salaries, and earnings are no exception to the general rule. But there are certain broad conclusions that cannot seriously be challenged. Perhaps the most important of these is that, since pre-war days, there has been a long-term trend whereby, in the sharing of the national cake, the position of salary earners has worsened relative to that of wage earners: white-collar workers (including those in the professions) have not been as successful as manual workers in obtaining increases in pay to match the steady fall in the value of money.

A Tighter Squeeze Ahead

This now familiar narrowing of differentials, the squeezing of the middle classes, is something which, in a general way, most of us recognize had to happen, and, however hard we may have been hit individually, there is a strong case for supposing that this redistribution of income has, on balance, been to the advantage of the community as a whole. But there is no clear evidence, as yet, of a reversal of this trend, and no one can forecast with any certainty how much tighter the squeeze may become. It is fairly clear that further increases in the cost of living are inevitable, even if we succeed in avoiding a runaway inflation. The battle of the different occupational groups to maintain or improve their economic position is bound to be fought with increasing bitterness and urgency.

What is the special importance in this connection of the doctors' present dispute with the Minister of Health? It is not just that the medical profession is an important middle-class group, whose success or failure in these pay negotiations will inevitably have repercussions in other closely related professions, such as dentistry. It is also—and this seems to many people much more important—that the doctors' claim raises the whole question of the pre-war position and the price changes since then in an acute form. The reason for this can be simply stated. When the National Health Service was created, the Spens Committee made their recommendations about doctors' pay in terms of 1939 money values. These figures had to be translated into money values for later dates, and the adjudication of Mr. Justice Danckwerts did this for 1948 and 1950-51. The doctors claimed, last July, an increase of 24% on these figures to take account of the rise in the cost of living since 1951. Official and unofficial spokesmen of other professions have, as a result of all this, been given what they regard as most useful ammunition in their fight to maintain their own standards.

The figures I am now going to quote are very rough, and are only for purposes of illustration. The Spens Committee, reporting in the late nineteen-forties, thought that the earnings of the average general practitioner ought to be, in terms of 1939 money values, around £1,100. As a result of the Danckwerts cost-of-living adjustment for 1950-51 this is now working out at perhaps £2,000: if the doctors' present claim were accepted this would be increased to nearly £2,500. That would mean an increase of 127% or so on the hypothetical 1939 figure.

Doctors and Civil Servants

Anyone in another profession who feels aggrieved about the way in which the middle-class squeeze has affected him has merely, therefore, to find a grade within his own profession with a 1939 salary somewhere in the £1,100 range, and show that the present salary for that grade is less than £2,500. He then feels justified, rightly or wrongly, in saying: 'Why should the doctors be allowed to have a cost-of-living adjustment for price changes since 1939 which, even if it is only partial, is much more favourable than anything my profession has been able to obtain?' Suppose, for example, that you are a higher civil servant. The pre-war starting salary of a male Assistant Secretary was £1,150; the present one is £2,000, including an increase granted within the last year. On the basis of these figures you might feel that your profession had only recently succeeded in obtaining pay adjustments equivalent to those the doctors had enjoyed since 1951.

Whatever the merits of the doctors' case (and they have, after all, a strong one), those involved in the handling of pay claims in the next few years in other professions will, because of these special features, be keeping an even closer watch on the outcome than they would normally do.

* The Listener (1957): **57**, 44 (10 Janaury). Reproduced by kind permission of the B.B.C. and of the author. This talk was given in the B.B.C. topical series 'At Home and Abroad'. The speaker had less than 24 hours notice and less than 4 minutes of broadcasting time in which to present his material.

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EDITORIAL

TREATMENT OF CANCER OF THE BREAST

Recent surgical opinion seems to throw some doubt on the wisdom of the advice we offer patients with carcinoma of the breast. Although it is traditional to insist that an early diagnosis be made and that the patient come ever earlier for treatment, it has been observed that the long-term results of our therapy have in many instances not justified the precipitancy with which we rush our patients into operation.

It appears that contrary to the usually accepted ideas, a patient who comes up for diagnosis after having 'neglected' the lump in her breast for some time, often carries a better prognosis than the patient who comes up immediately the lump is felt. This apparent anomaly is explained on the basis that it is the relatively slow-growing tumours which do best and that a tumour which is quickly noticed because it grows quickly carries a bad prognosis, whether the treatment is instituted early or late. If the prognosis is dependent on the nature of the tumour then early treatment may have very little effect on the ultimate result. This is dangerous teaching because it may lead to giving wrong advice and cause patients to neglect strange lumps which may appear, and so certain cases which might have obtained a permanent cure as a result of operation may miss their opportunity. It can still be categorically stated that the sooner the cancer is out, the better.

Another disturbing feature in our operative therapy for breast cancer is its mutilating nature, which we are not able to mitigate for the patient who comes early. There is no reward for her in this respect, nor can we promise her a better prognosis.

Halsted's operation is a mutilating procedure with a very far-reaching psychological effect. Women don't forget it, and their character and personality are often scarred as well as their bodies. If there were a way to avoid this very extensive surgery it should be considered against this background. Within the last few years McWhirter, in Edinburgh, has been running a series of cases of carcinoma of the breast in which he has used irradiation of the axilla combined with local mastectomy. McWhirter's results have recently been subjected to a close analysis by Ackerman,[1] whose conclusion is that, if patients are properly selected and are operated on by trained surgeons, radical mastectomy should yield a

VAN DIE REDAKSIE

BEHANDELING VAN BORSKANKER

Dit skyn of onlangse chirurgiese mening 'n mate van twyfel werp op die wysheid van die raad wat ons aan pasiënte met borskanker bied. Alhoewel dit tradisioneel is om daarop aan te dring dat 'n vroeë diagnose gemaak moet word en dat die pasiënt steeds vroeër vir behandeling moet kom, is daarop gelet dat die langtermyn resultate van ons geneeskundige behandeling in baie gevalle nie die oorhaastigheid waarmee ons op pasiënte opereer, regverdig nie.

In stryd met die algemeen-aangenome idees, skyn dit of 'n pasiënt wat, vir 'n diagnose kom, nadat sy die klont in haar bors vir 'n geruime tyd ‚verwaarloos' het, dikwels 'n beter vooruitsig het as die pasiënt wat kom sodra sy die klont in haar bors voel. Hierdie skynbare afwyking word verklaar op die grondslag dat dit die betreklik stadig-groeiende gewas is wat die beste daartoe is, en dat 'n gewas wat gou bemerk word omdat dit vinnig groei, 'n slegte prognose inhou, al word die behandeling ook al vroeg of laat begin. Indien die prognose afhang van die aard van die gewas, mag vroeë behandeling weinig uitwerking op die uiteindelike resultaat hê. Dit is 'n gevaarlike leer om te verkondig aangesien dit aanleiding gee tot verkeerde advies en veroorsaak dat pasiënte snaakse klonte wat mag verskyn, mag verwaarloos, en op hierdie wyse verloor sommige pasiënte, wat 'n permanente kuur as gevolg van 'n operasie kon gehad het, hulle kans. Kategories kan dit verklaar word dat hoe gouer die kanker verwyder word, hoe beter.

'n Ander verontrustende kenmerk van ons operatiewe terapie van borskanker is die skendende aard daarvan, waaraan ons nie in staat is om iets te doen om dit minder skendend te maak vir die pasiënt wat vroeër kom nie. Dit hou geen beloning vir haar in nie, en ons kan haar ook nie 'n beter vooruitsig belowe nie.

Halsted se operasie is 'n skendende metode met baie verreikende psigiologiese gevolg. Vrouens vergeet dit nie en dikwels word nie alleen hulle liggame geskend nie, maar ook hulle karakter en persoonlikheid. Indien daar 'n wyse bestaan om hierdie baie ernstige chirurgie te vermy, behoort dit teen hierdie feit oorweeg te word. Binne die afgelope paar jaar het McWhirter, in Edinburg, 'n reeks van gevalle van borskanker behandel waar hy bestraling van die okselholte tesame met lokale borsverwydering gebruik het. McWhirter se resultate is onlangs onderwerp aan 'n sorgvuldige ontleding deur Ackerman,[1] wat tot die gevolgtrekking gekom het dat, as pasiënte behoorlik uitgesoek word en deur opgeleide chirurge geopereer word, radikale borsverwydering 'n groter aantal permanente kure behoort op te lewer. Daar is egter nie veel daar toe nie en dit mag wees dat McWhirter

greater number of permanent cures. There is, however, very little in it, and it may well be that McWhirter's treatment of cancer of the breast should be followed elsewhere, but unfortunately the matter is not so simple as all this. Radical mastectomy is a fairly standardized procedure which can be performed by a great number of surgeons of reasonable training and ability all over the world, and their results will be closely comparable; but McWhirter's treatment demands more than average radio-therapeutic ability and equipment. A radical mastectomy can be performed anywhere, whereas proper irradiation demands admission to specialized centres with highly-skilled personnel. In McWhirter's hands the results of the Halsted operation can probably be equalled, but there is no possibility of this where less than adequate radiotherapy only is available. Working on the principle of the greatest good for the greatest number, it appears that the time has not yet come to recommend McWhirter's method for general use in carcinoma of the breast. Halsted's operation is still our sheet anchor and the safest manoeuvre generally available. In spite of statistics there is no doubt that, for the individual case, the sooner the carcinoma is removed the better.

We have no way of distinguishing the case that is going to do well from the case that will not do well, and although we are well aware that an operation is no ideal therapy, the disease will not wait on the discovery of this ideal. We can only offer our patients the best treatment available *at the moment*. With the recent advances in knowledge of carcinoma of the breast, with the hope that we are on the brink of great discoveries, it becomes more and more important to keep the patient going somehow. When the moment comes, the great discoveries and the ideal forms of treatment will only be available to the survivors. We cannot deal with statistics, our work is with individual human lives.

1. Ackerman, L. V. (1955): Cancer, **8**, 883.

op ander plekke gevolg behoort te word, maar ongelukkig is die saak nie heeltemal so eenvoudig nie. Radikale borsverwydering is 'n taamlik gestandardiseerde metode wat deur 'n groot aantal chirurge met 'n redelike opleiding en bekwaamheid oor die hele wêreld uitgevoer kan word, en hulle resultate sal baie min verskil; maar McWhirter se behandeling vereis meer as gemiddelde radio-terapeutiese bekwaamheid en toerusting. 'n Radikale borsverwydering kan oral uitgevoer word, terwyl behoorlike bestraling toelating tot gespesialiseerde sentrums met deeglik-geskoolde personeel vereis. In McWhirter se hande kan die resultate van die Halsted-operasie waarskynlik geëwenaar word, maar daar is geen moontlikheid hiervan waar minder as doeltreffende radio-terapie alleen beskikbaar is nie. Werkende op die prinsiep van die grootste voordeel vir die grootste getal, skyn dit dat die tyd nog nie daar is om McWhirter se metode vir algemene gebruik by borskanker aan te beveel nie. Halsted se operasie is nog ons noodanker en die veiligste maneuver wat algemeen beskikbaar is. Ten spyte van statistieke, bestaan daar geen twyfel nie dat, sover dit die individuele geval betref, hoe gouer die kanker verwyder word, hoe beter.

Daar is geen wyse waarop ons kan onderskei tussen die geval wat goeie resultate gaan oplewer en die wat nie goeie resultate gaan oplewer nie en alhoewel ons bewus is dat 'n operasie nie die ideale geneeskundige behandeling is nie, sal die siekte nie wag totdat ons hierdie ideaal bereik nie. Ons kan slegs ons pasiënte die beste behandeling aanbied wat *op die oomblik* beskikbaar is. Met die onlangse vooruitgang van ons kennis van borskanker, met die hoop dat ons op die vooraand van groot ontdekkings staan, word dit al hoe belangriker dat ons die pasiënt op een of ander wyse aan die gang hou. Wanneer die oomblik aanbreek, sal die groot ontdekkings en die ideale wyse van behandeling slegs aan die oorlewendes beskikbaar wees. Ons kan nie met statistieke handel nie, ons werk is met individuele mense-lewens.

1. Ackerman, L. V. (1955): Cancer, **8**, 883.

POLIOMYELITIS VACCINE BY MOUTH

A second report has come from America confirming the development of an immune response in subjects given live attenuated poliomyelitis virus by mouth.[1] In all cases investigated, the precaution was taken of giving immune serum-globulin intramuscularly, which probably reduces the risk of producing paralytic poliomyelitis,[2] although it prevents neither the development of an antibody response nor intestinal carriage of the virus. In this report, Koprowski and his co-workers describe a series of 24 infants, aged 10 days to 6 months, who were given large doses (1,000-100,000 infective doses) of type-1 and type-2 virus in their milk. All developed an active immunity after infection—itself a completely asymptomatic process—and the investigators claim that it has now been shown conclusively that the oral administration of live attenuated poliomyelitis virus is perfectly safe. Before this series, other trials had been reported with similar findings in adolescents and adults.[3] Koprowski *et al.* hope that long-term follow-up of their infant series will show that the level of induced homotype antibodies has conferred permanent protection.

A practicable method of oral immunization has long been awaited, and these results are therefore of the utmost interest. Koprowski *et al.* at the Lederle laboratories appear to be the only workers actively engaged in this project and, although Dick of Belfast has begun to test the antigenic power of Koprowski's vaccine,[4] the only information on the subject so far has come from Koprowski's own laboratory. From February 1950 until 1956, 155 subjects (mostly children, but also adults) were given a live attenuated poliomyelitis virus (of a type to which they possessed no antibodies) in diluted milk or in a gelatin capsule.[5] They were then quarantined and submitted to rigid clinical observation. None developed any signs of illness, and no virus was isolated from the blood-stream or from throat swabs of these persons —and all developed antibodies. However, most cases—and constantly those fed upon the type-1 poliomyelitis virus—excreted the virus in the faeces for considerable periods, some up to 108 days. It is upon this issue that the whole matter hinges.

Is the discharge of attenuated polio virus dangerous?

If the attenuated virus can be relied upon not to produce the disease in a harmful form on infecting another person, and if the strain can also be relied upon not to regain its virulence, then the excretion of the virus by the vaccinated subjects need cause no alarm—indeed it might lead to the immunization of those accidentally infected. Vaccination against smallpox is a partly analogous case; we know that although the vaccinated person discharges attenuated smallpox virus (in the form of cowpox virus) this does not give rise to human smallpox under any conditions. It would appear that the future of oral vaccination with live attenuated polio virus depends on the answer to the question whether there is any danger in its indiscriminate discharge by the immunized person. Unless the answer is definitely negative the problem remains for the present unsolved. Koprowski believes the future of live virus immunization to be bright. 'The number of immunizations of non-immune individuals without accident has given increased confidence in the use of live attenuated strains', he writes.[6]

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BENIGN PULMONARY HISTOPLASMOSIS (CAVE DISEASE) IN SOUTH AFRICA

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The literature of histoplasmosis in Africa contains references to 23 cases of the systemic and of the localized cutaneous forms of the disease.[1–18] Five of the cases were diagnosed in England[2, 5, 13, 16] but the patients had spent some part of their lives on the African continent and probably acquired the infection there. The literature also refers to the isolation of a strain of the organism in Africa which, in the opinion of the authors, differs sufficiently from the classical form to justify a new type-name (*H. duboisii*),[19] and to several surveys[20, 21] in which the histoplasmin positivity rate has been shown to vary from 0·0% to 17·8% in different parts of the continent. Nowhere however in the African literature is reference made to the benign pulmonary form of the disease which has been described in the American literature under a variety of names such as cave disease, angle-worm pneumonitis, 'unusual pulmonary disease', acute miliary pneumonitis, and granulomatous pneumonitis.

Our attention was first drawn to this form of the disease in South Africa by the development of acute pneumonitis in 3 students 14 days after they had explored deep limestone caves in the Potgietersrust district, Transvaal. Although we failed to isolate *H. capsulatum* from the cases we pursued our investigations along 4 main lines:

1. By contacting all past and present members of the Transvaal Spelaeological Society and obtaining from them a complete medical history together with X-rays of the chest and an investigation of their histoplasmin sensitivity. Suitable control groups were investigated along similar lines.

2. By observing all new members of the Society before and after their initial visits to suspected caves.

3. By attempting to isolate *H. capsulatum* from the soil, atmosphere and fauna of the caves in the Transvaal.

4. By exposure of experimental animals in caves in which human beings contracted the disease.

The results of these investigations form the subject of this communication.

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Epidemiology

After the first description of histoplasmosis by Darling in 1906[22] and his observation of the causative organism, the only references to the clinical manifestations of the condition over the next 40 years were to a fatal systemic form.[23] In 1945, however, Palmer,[24] investigating pulmonary calcification in student nurses, found a correlation with histoplasmin sensitivity. As a result of his findings he suggested that a benign form of histoplasmosis was widely prevalent in certain American states. Subsequent surveys confirmed his postulate and showed that histoplasmin sensitivity, indicating past or present contact with the causative organism, was widespread in the USA.[25] These surveys made it clear that, far from being a uniformly fatal condition of infrequent occurrence, histoplasmosis was, in certain states of the USA, a common infection from which the vast majority of patients completely recovered. The highest incidence appeared to be in the states bordering the Mississipi river. In some areas of Ohio it was found to be as high as 86·4% amongst student nurses.[26] In 1953 Furcolow and Grayston[27] reported a series of epidemics of disseminated pneumonitis and showed them to be due to infection with *H. capsulatum*. The epidemic form was shown to occur in association with enclosed spaces such as caves,[28] silos,[29] underground storm-shelters,[30] pigeon lofts,[31] chicken runs[32] and other situations suitable for the saprophytic growth of the fungus.

The occurrence of the 3 cases to which we have referred suggested to us that benign pulmonary histoplasmosis might occur in South Africa and our subsequent investigations have proved this to be so.

EPIDEMIOLOGY IN SOUTH AFRICA

As a preliminary step histoplasmin skin-tests were carried out in 4 groups of subjects. The results are shown in Table I. The student group was chosen on the basis of having had no experience of spelaeology but in other respects such as sex, age and socio-economic status being comparable to the spelaeologist group. Those designated as Group A of the laboratory workers were drawn from

TABLE I. RESULTS OF HISTOPLASMIN SKIN TESTS (1/1000)

	Positive	%	*Negative*	%	*Total*
Students	—	—	39	100	39
SAIMR Lab. Staff (Group A) ..	—	—	25	100	25
SAIMR Lab. Staff (Group B) ..	13	12	93	88	106
Spelaeolgists, Cape*	—	—	20	100	20
Spelaeologists, Transvaal	53†	94·5	3	5·5	56
Clinical Cases ..	46	100	—	—	46

Positive = 5 mm. induration at 48 hours.

* The absence of clinical cases and histoplasmin sensitivity in the Cape section of the South African Spelaeologists Society is discussed in the text.

† 7 spelaeologists gave no history of illness (4 histoplasmin-positive and 3 histoplasmin-negative).

the staff at the serum farm of the South African Institute for Medical Research, where no mycological laboratory work has ever been carried out and which is situated 7 miles from the central laboratory of the Institute. Group B comprised workers in the central laboratory, in which a medical mycology department is situated and where strains of *H. capsulatum* have been maintained in culture for many years.

The striking difference in positivity between students, laboratory workers and spelaeologists strongly suggests that the underlying factor is contact with histoplasma, which was common amongst the spelaeologists, unknown to the students, and, in the laboratory workers, depended upon whether or not they had worked at the central laboratory, where *H. capsulatum* cultures are maintained. The histoplasmin-sensitivity tests in spelaeologists show 94·5% positivity. Investigation of the medical history reveals, in 82·1% of them, a story consistent with the occurrence of benign pulmonary histoplasmosis some 5-18 days after their first experience of cave exploration. The histories obtained showed that the condition occurs in epidemic form whenever a new group joins the Society. Typical histories of such epidemics are as follows:

Epidemic 1. On 12 March 1953 4 students explored the ramifications of a limestone cave system in the Potgietersrust district. One of the students proceeded only part of the way into the caves before returning to the surface. The other three continued until, after some hundreds of yards, they reached a dead end. The floor was covered with a deep layer of bat guano and they were exposed to the dusty atmosphere for several hours. Fourteen days later each of the three developed acute pneumonitis, from which they recovered after a period of 3 weeks. This formed the first group of such cases to which our attention was drawn. The 4th student, who had spent only a short time in the cave, developed no clinical signs, but all four became histoplasmin-positive.

Epidemic 2. On 26 April 1953 4 adult Europeans decided to explore a large limestone cave in the Thabazimbi area with a view to deciding whether it was suitable for mining bat guano as fertilizer. They spent 3 hours in the cave digging through guano at different points to determine its depth. In doing so they exposed themselves to inhalation of the dust-laden atmosphere. Seven days later all four developed typical attacks of pneumonitis, from which they completely recovered after a period of several weeks. After the illness all became histoplasmin-positive.

Epidemic 3. On 16 July 1953 7 students explored a limestone cave leading off a 70-foot pothole. Few bats were observed and the depth of bat guano was slight. Not much digging was done but the caves were dusty in the dry portions. The students spent the greater part of the day in the caves. After 9-14 days all developed attacks of typical pneumonitis, from which they recovered within 3-14 days.

The appearance of an acute respiratory infection 5-18 days after spending some hours in cave exploration was encountered in many other subjects whom we contacted through the Transvaal Spelaeological Association. The subjects could be divided into 3 groups:

Group 1. In 29 persons, comprising the first group, exposure had occurred some weeks or even years previously and the diagnosis was established in retrospect in 26 of them. There were 22 in whom there was a clear-cut history of illness following exposure in an infected cave and in whom the histoplasmin skin-test was positive. Four others gave a positive skin-test but no history of illness although they had explored infected caves. Three were negative reactors, one of whom was a most active spelaeologist and probably had had more experience in cave exploration than any of the other members. She accompanied most of the members of the group during the expeditions after which they developed their illness and yet at no time has she given a history of illness resembling cave disease and has remained histoplasmin-negative to date.

Group 2 comprised 10 subjects who were first observed during or shortly after the acute stage of their illness. They showed, at this stage, doubtful or negative histoplasmin skin-tests but some weeks or months later were found to be definitely histoplasmin-positive.

Group 3. The third, and most significant, group was composed of 14 new members of the Society who gave no history of ever having been in caves and on whom histoplasmin skin-tests were carried out as soon as they joined the Society. At the same time their chests were X-rayed, a full blood-count was carried out, and their blood serum was examined for antibodies against rickettsiae and various viruses, including psittacosis. They were kept under observation throughout their activities with the Society and it was found that in each instance an attack of pneumonitis followed exposure in a cave known to be infected. A change from histoplasmin-negative to histoplasmin-positive followed the illness and, in most instances, variable radiological changes were found in the lungs during the acute phase.

It became apparent, as the investigation progressed, that not all cavers were infected and that the severity of the illness was directly related to the length of exposure. In some instances new members of the Society explored caves with impunity but subsequently developed infection after exploring some other cave system. Gradually it became possible to determine those caves in which it did not appear to occur. One cave in particular, known as Johnson's Pothole, Hennops River, (Fig. 1) uniformly led to infection of subjects who had not previously suffered from the disease, with the exception of the 3 members to whom reference has already been made. Fig. 1 shows vertical and horizontal plans of the cave, from which it will be seen that entry was gained by a vertical shaft from the foot of which it is necessary to crawl along a narrow tunnel until it becomes possible to climb down into the main cavern. During the course of the investigation members of the Society who had not proceded beyond point A became infected, but the majority explored the main cavern and further ramifications. Experimental animals to which reference will be made later were exposed at point C.

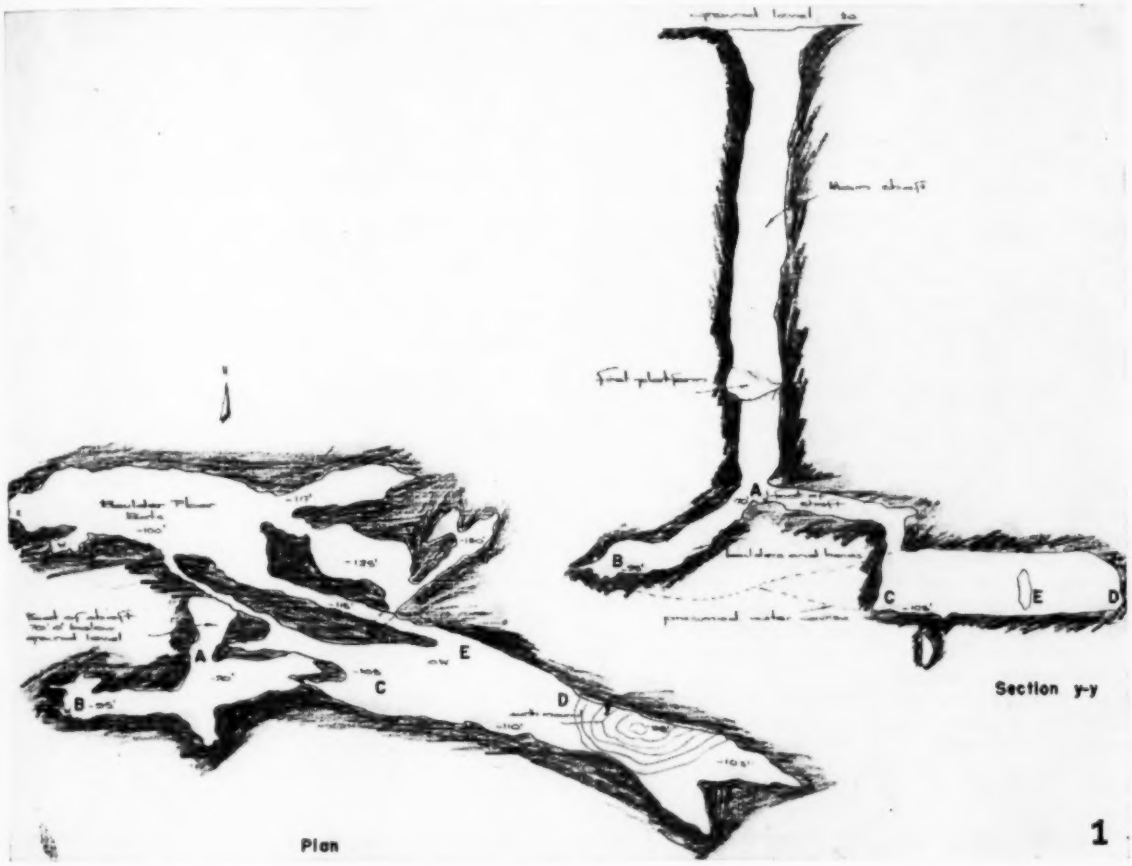

Fig. 1. Plan and section of Johnson's Pothole, Hennops River, Transvaal. Scale 1 inch = 25 feet. J. M. H.

It also became clear that once subjects had suffered from an attack of the disease they did not again develop it even upon repeated exposure in infected caves. In the course of discussion with members of the Spelaeological Society it became apparent that a history of cave disease was confined to those in the Transvaal. No history of cave disease was obtainable from the Cape section of the Society.

At our request histoplasmin skin-tests were carried out by Dr. R. L. Kleinman on 20 members of the Cape Spelaeological Society. No positive results were obtained and no history of any illness resembling cave disease was elicited. Only 3 of the Cape members had visited caves in the Transvaal, and in each instance it was to a cave in which, in our experience, the probability of infection is low.

CLINICAL AND RADIOLOGICAL FEATURES

All cases which came under our observation, or in which a past history of infection was obtained, followed a uniform pattern of illness varying only in severity. Between 5 and 18 days after exposure the patients

complained of lassitude, pains in the limbs and joints, backache, headache, pyrexia, coryza, and non-productive cough. Dyspnoea was a common symptom and in some cases persisted after the acute attack. In severe cases rigors were not uncommon.

Radiological examination during the acute stage of the illness showed a variable picture. The pulmonary changes which occurred in 19 cases could be divided into 4 distinct groups. A widespread miliary nodulation was shown by 7; 7 presented with a patchy loss of translucency similar to the changes seen in a virus pneumonitis; 3 had coarse ill-defined, pea-sized nodules which, in one case, were confined to the right upper lobe and in the other two were diffusely scattered; 2 cases showed a generalized increase in broncho-vascular markings, which might have been passed as normal had the cases not been followed up and shown to have cleared completely. No radiological changes were observed in some of the cases. The fleeting nature of the radiological features and the difficulty of obtaining more than one or two plates during the acute phase of non-hospitalized cases may partly explain the negative findings in some cases.

Despite radiological and clinical evidence of pneumonitis physical signs on auscultation of the lungs were remarkably few. Even in the most severe cases they seldom amounted to more than an occasional crepitation and rhonchus. No hepatomegaly, splenomegaly or enlarged lymph-glands were found in any of the cases. Depending upon the severity of the attack, the acute stage of the illness lasted from 1 to 21 days, after which recovery was complete although convalescence was often prolonged in the severe cases. The severity of the attack was directly related to the length of exposure. The most severe cases had spent not less than 6 hours in the infected caves. The only significant difference between the cases under our observation and those described in the American literature[36] was the absence of pulmonary calcification. This may, however, be due to the fact that the longest follow-up period in any case under our personal observation from the time of the acute phase (groups 2 and 3) is 3½ years. One of the cases diagnosed in retrospect (group 1) occurred 8 years before coming under our observation and 4 of them 5 years before, but they also showed no evidence of calcification. The significance of this feature is obscure but will be the subject of further investigation.

Laboratory Findings

Haematological examination yielded no consistent pathological changes but occasionally there was a slight leucocytosis and an increase of polymorphonuclear cells. The sedimentation rate was increased during the acute phase of the illness. Serological tests for typhoid, paratyphoid and brucellosis were uniformly negative. Complement-fixation tests were carried out for rickettsial infections and for virus infections but no diagnostically positive titres were obtained. Skin tests with 1/1,000 dilution of histoplasmin before the onset of the illness in unexposed persons were uniformly negative (group 3) and were negative during the early stages of the acute illness (groups 2 and 3). Between 4 and 8 weeks after the onset of the infection the histoplasmin skin test usually became positive and the strength of the reaction increased to a maximum which, as a rule, was reached between the 8th and 10th week after the onset of the acute phase. In some cases the change to histoplasmin-positivity was delayed. In 10 cases histoplasmin complement-fixation tests were carried out during the acute stage of the illness; positive results were obtained in 6. Dr. M. L. Furcolow carried out 4 of the tests and the remainder were made by us with antigen kindly supplied by Dr. K. W. Walls. The results of the skin tests carried out with other fungal and bacterial extracts are shown in Table II. Numerous attempts to isolate *H. capsulatum* from the sputum of patients were uniformly negative.

TABLE II. FUNGAL ANTIGEN SKIN-TESTS IN SOUTH AFRICAN SPELAEOLOGISTS

Extract	*Dilution*	*Negative*	*Positive*	*Total*
Histoplasmin ..	1/1000	3	53	56
Coccidioidin ..	1/100	53	—	53
Tuberculin	1/5000	34	19	53
Haplosporangin ..	1/100	21	—	21
Trichophytin ..	1/10	8	1	9
Blastomycin ..	1/100	8	—	8
Monilia (killed organisms)		2	1	3

CASE HISTORIES

A typical case history from each of the three groups is set out as follows:

Group 1

C.W.R., European male aged 27, was first seen by us on 27 August 1954. In 1951, in company with several others, he had explored Johnson's Pothole. Later we were able to make contact with 5 other members of the party, all of whom gave a similar history. C.W.R. developed pyrexia and rigors 14 days after his visit to the cave. He was confined to bed for 8 days and his case was diagnosed as influenza with bronchitis and pneumonia. No X-rays were taken. No further history was obtainable except that the patient said it was 3 or 4 weeks before he recovered and he had no relapse. When we saw the patient 3 years later all laboratory investigations and an X-ray of the chest were negative. Skin tests with coccidioidin, haplosporangin and blastomycin were negative. Histoplasmin and tuberculin skin-tests yielded strongly positive results. A total of 22 similar cases with histories varying only in detail were found during the course of our investigations.

Group 2

W.K.H., a European male aged 22 years, was a medical student. He became acutely ill 14 days after exploring a cave system in the Potgietersrust district. He developed pyrexia up to 105°F accompanied by severe rigors and drenching sweats, severe throbbing headaches, and pains in the muscles and joints, particularly in the back. Throughout the illness the patient had a non-productive cough and complained of pleural pain upon deep inspiration. No splenomegaly, hepatomegaly or lymphadenopathy was found. A striking feature of the patient's condition, and one which was found in several others who were acutely ill, was a negative mental attitude leading to non-cooperation during, and for some time after, the acute stage of the illness.

X-ray of the chest showed a widespread coarse miliary mottling tending to coalesce towards the hilar regions; there was no evidence of hilar glandular enlargement (Fig. 2). Three weeks later the lung fields were clearing and the lesions were becoming smaller and more discrete (Fig. 3). Four months later the patient was again X-rayed and showed a completely normal appearance with no calcification (Fig. 4).

Despite the extensive X-ray changes physical signs in the chest were minimal. Laboratory investigations yielded the following results: Hb. 17; rbc 5·8; wbc 7·0 (neutro 55·4%, mono 8·2%,

Fig. 9.

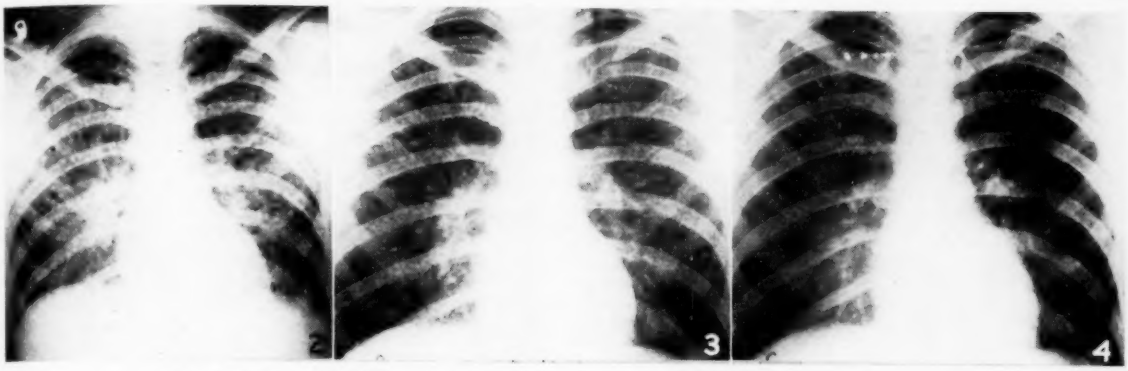

Fig 2. WKH. Coarse miliary mottling of lung at onset of illness and 14 days after exposure.
Fig. 3. WKH. Clearing of lung fields, with lesions becoming smaller and more discrete.
Fig. 4. WKH. Complete clearing of chest 4 months later. No calcification.

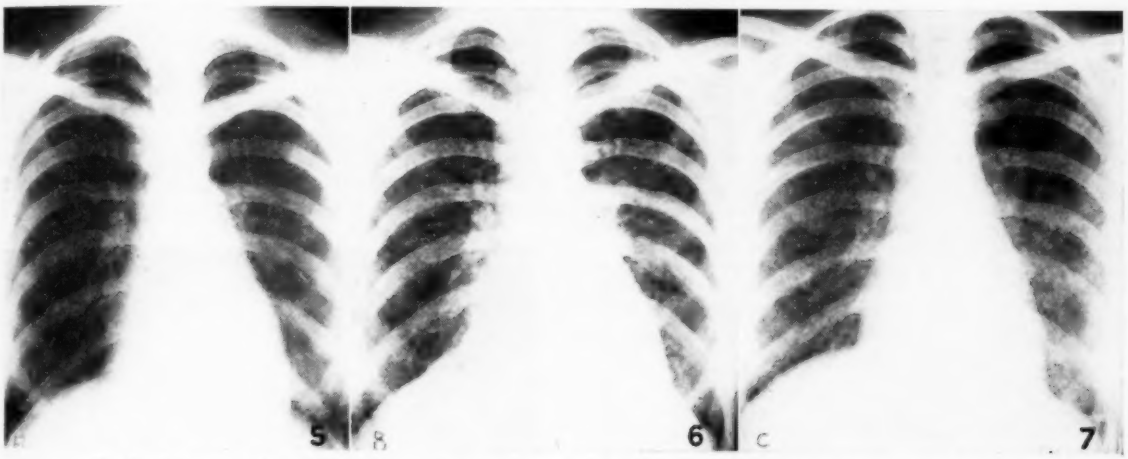

Fig. 5. P.V. First day of illness. *Fig*. 6. PV. Vague miliary appearance of chest on 5th day of illness.
Fig. 7. PV. More definite miliary appearances on 7th day of illness.

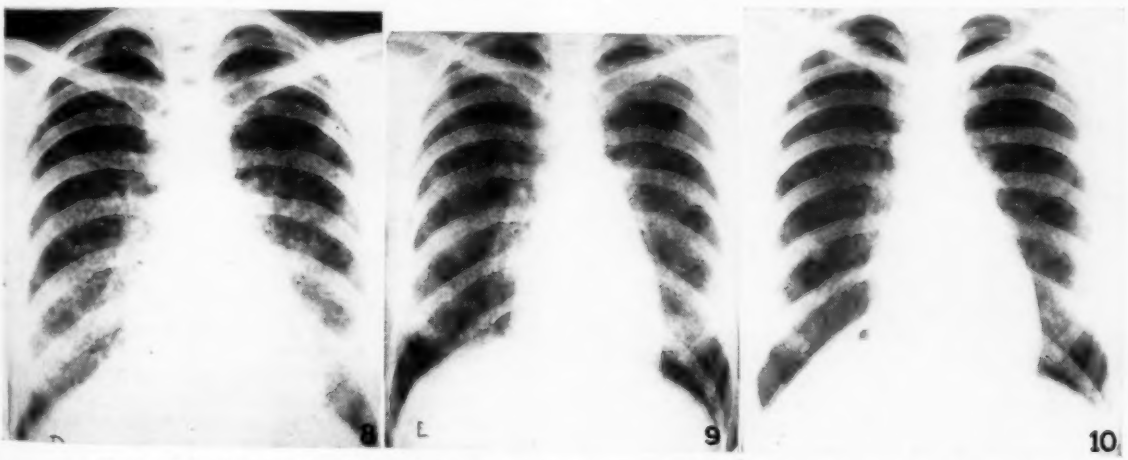

Fig. 8. PV. Increase in number of miliary nodules, with tendency to coalescence, on 14th day of illness.
Fig. 9. PV. Clearing of lung fields on 21st day of illness. *Fig*. 10. PV. Showing normal X-ray of chest 6 months after illness.

ympho 32·8%, eos 2·5%). ESR (Wintrobe) 22 mm. in 1 hour (marked increase). Agglutination tests for typhoid and paratyphoid were negative, rickettsial complement-fixation tests and virus complement-fixation tests (psittacosis, mumps, herpes, and lymphocytic choriomeningitis were negative. Blood culture yielded no growth. No pathogens were isolated from the scanty specimens of sputum obtained. The histoplasmin skin-test on 7 March 1953, 2 weeks after the onset of illness, was doubtful. A month later it was weakly positive. Eleven months later it was definitely positive. A similar course was followed in the other cases seen for the first time during the acute stage of their illness.

Group 3

P.V., a European female aged 21 years, first came under our observation when she had had no experience of cave exploration. Her histoplasmin skin-test, carried out on 5 May 1953, yielded a negative result. On the 24th of the same month she explored Johnson's Pothole with 2 other members of the Society, each of whom had had a previous attack of cave disease and neither of whom developed any illness on this occasion. Twelve days later the patient complained of a sore throat and felt unwell with headache, backache and aches and pains in the muscles and chest. She was found to have a temperature of 102°F. Throughout the illness she had an unproductive cough and developed slight dyspnoea, which continued for some time after the acute phase of the illness was over. No pleural pain occurred and chest signs were minimal. Radiological examinations were carried out at regular intervals throughout the illness and until complete recovery. An X-ray plate of the chest taken on the first day of illness (5 June), was negative (Fig. 5). Four days later, she was again X-rayed and a vague miliary appearance was noted (Fig. 6). Two days later the miliary appearance became more definite (Fig. 7), and after an interval of a further 7 days the nodules became more numerous, with a tendency towards coalescence towards the bases (Fig. 8). A radiograph taken after another 8 days (21 days from the onset of symptoms) showed a marked clearing of the lung fields, the lesions having become smaller and more discrete (Fig. 9). A follow-up radiograph 6 months later showed a completely normal picture (Fig. 10) with no evidence of calcification. No enlarged glands could be detected at any time.

A total of 14 cases similar to this came under our observation during the period of investigation.

Isolation of the Fungus

Attempts to isolate *H. capsulatum* were directed along 3 main lines:

1. Culture and animal inoculation of sputum from clinical cases.
2. Culture and animal inoculation of samples of soil and air filtrates, and of the viscera and intestinal contents of bats from known infected caves.
3. Exposure of experimental animals in infected caves.

All attempts to isolate the organism from sputum and blood from clinical cases were uniformly unsuccessful.

A large number of samples were taken from various parts of many caves and treated by the method recommended by Larsh *et al.*[33] When it became apparent that the individuals entering Johnson's Pothole invariably became infected, investigations were mainly directed to this cave. In addition to the samples of soil examined in our own laboratories, soil was sent to Dr. Emmons of the Laboratory of Infectious Diseases, Bethesda, for the same purpose. A large number of soil samples was examined but no positive results were obtained. With the use of a small air-pump and millipore filters several attempts were made to isolate the fungus from the atmosphere of the caves. This also proved abortive. Cultures and animal inoculations from the intestine, spleen, liver, and lungs of several bats caught in the caves yielded no fungal growth.

Culture plates were exposed to the atmosphere in some of the infected caves for varying periods of time. They yielded many different fungi but no *H. capsulatum* was isolated by this method.

On 4 March 1956 7 members of the Transvaal Speleological Society, including one of the authors (JFM), explored Johnson's Pothole to collect specimens of soil and to attempt to demonstrate that the infection was air-borne. Two of the 3 members who had not previously been in caves wore respirators while the other new member enjoyed no such protection. Each of the 3 new members was histoplasmin-negative, and gave a negative X-ray plate of the chest. Of the remaining 4 members 3 were histoplasmin-positive and had past histories of the infection. The 7th member of the group was histoplasmin-negative, but had explored caves on many occasions and had never contracted the illness.

No illness of any kind developed in the 4 experienced members of the group but the unprotected new member, who wore no respirator, developed a typical acute attack of pneumonitis 14 days after exposure and after 4 weeks became histoplasmin-positive. The two new members who wore respirators developed so slight an illness 14 days after exposure that, had they not been under constant observation, it would not have been noted. On

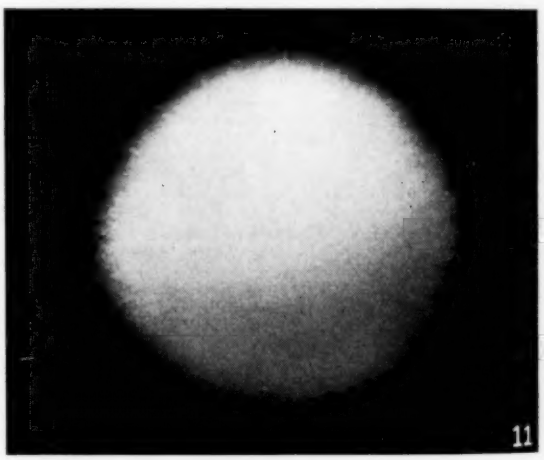

Fig. 11. Culture of *H. capsulatum* from test mice.

the 14th day each felt slightly unwell, appearing to be developing a cold in the head and then, in less than 24 hours, had completely recovered. Some weeks later their histoplasmin skin-tests were found to be positive. The respirators were not of the closed-circuit type and, as they fitted rather poorly at the nasolabial fold, it is felt that some air was inhaled which had not passed through the filter pad. In view of the mild nature of the illness and the subsequent change from a histoplasmin-negative to histoplasmin-positive skin-test it is felt that they suffered from a very mild infection comparable to that

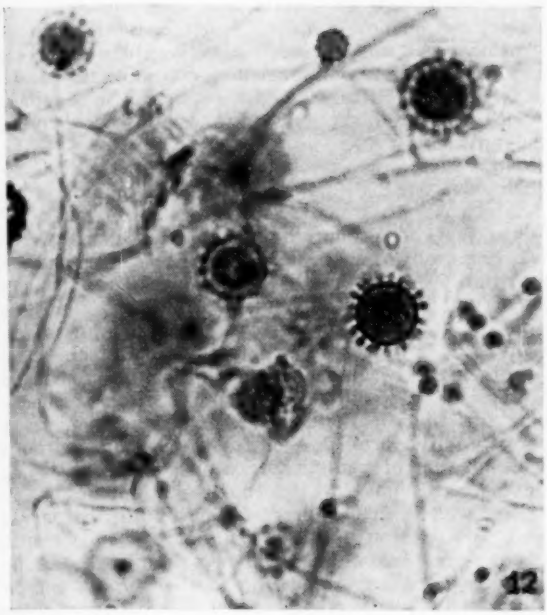

Fig. 12. Microscopic apearance of culture shown in Fig. 11.

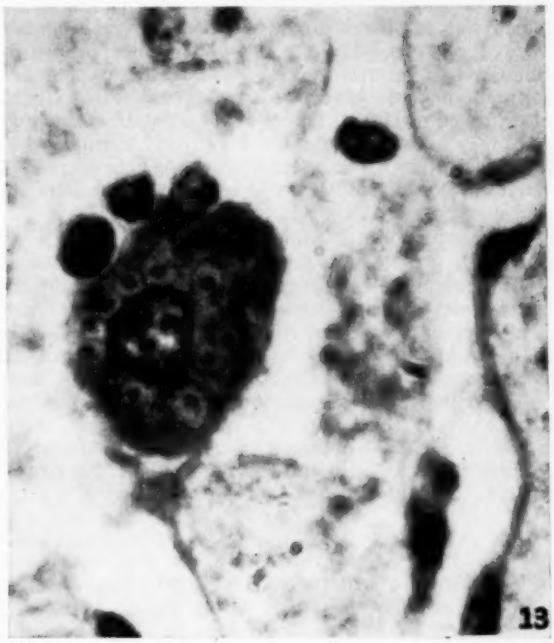

Fig. 13. Section of liver of mouse from which the culture shown in Figs. 11 and 12 was obtained.

which develops in subjects who are exposed for a very short period, although in this instance they had actually been in the caves for 6 hours. This suggestion is confirmed by the fact that both patients have since exposed themselves without respirators in Johnson's Pothole and neither developed any evidence of cave disease. The filter pads from both respirators were soaked in normal saline and the saline extract injected intraperitoneally into white mice. Mice injected with the extract from one of the filter pads developed histoplasmosis and the organism was successfully isolated from the spleen and liver of the animals. (Fig. 11, 12). Sections of the livers of the infected animals showed typical yeast bodies in the Kupfer cells (Fig. 13).

On 9 September 1956 5 members, including one of the authors (JFM), took animals into Johnson's Pothole. The experimental animals consisted of monkeys, rabbits, guinea pigs, rats, and mice. Half of the mice were weanlings. The animals were taken to the lowest level of the cave, where they were exposed to the dust-laden atmosphere for a period of 5 hours.

After returning to the laboratory the animals were kept under observation. Histoplasmin skin-tests were carried out on surviving monkeys, guinea pigs and rats at 14 day intervals until the 12th week after exposure. All gave negative results. Animals from each group were killed from the first day onwards and full post-mortem examinations performed. The following investigations of the lung, liver and spleen were carried out: (1) Macroscopic examination, (2) histopathological examination, (3) culture for fungi, bacteria and virus, and (4) passage of lung tissue through white mice.

From the 2nd day onwards the lungs showed

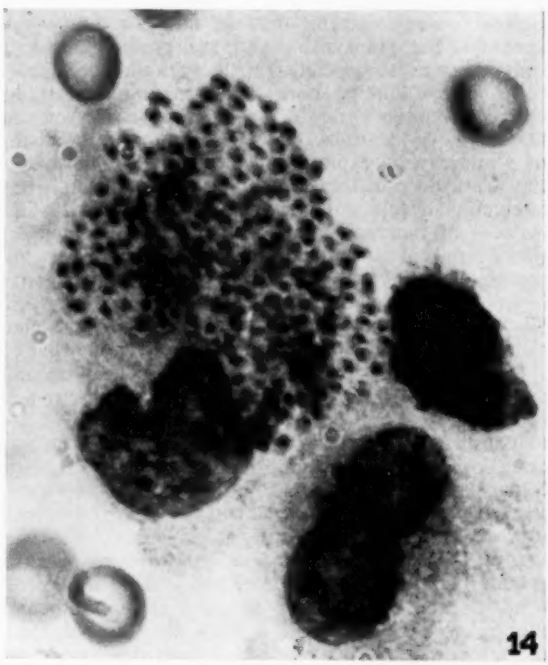

Fig. 14. Splenic smear from mice through which lung tissue of experimental animals had been passaged.

macroscopic evidence of broncho-pneumonia, a finding which was confirmed upon histopathological examination of sections. Despite the pneumonic findings no fungi were observed in PAS-stained sections and cultures for bacteria yielded no pathogens. Virus studies also proved negative. Direct cultures for *H. capsulatum* were positive. Passage of the lung tissue through white mice yielded positive splenic smears (Fig. 14). The fungus was recovered from at least one of each of the species exposed in the caves.

The results of the animals experiments will be reported in greater detail in a further communication.

The features of the fungal strains recovered from the experimental animals were identical with those from 5 fatal clinical cases of histoplasmosis reported from South Africa[3, 9, 18] and with classical strains of *H. capsulatum*. No characteristics of *H. duboisii* were observed in animal tissues or in the cultures obtained from them. Re-examination of sections from the 5 fatal systemic cases observed by us in South Africa[3,9,18] confirm that they also showed none of the features of *H. duboisii*.

DISCUSSION

In 1942 Simson and Barnetson[3] reported a fatal systemic case of histoplasmosis from South Africa. In 1951 Murray and Brandt[9] reported 3 more such cases and Lurie and Brandt[18] observed a 5th case. The origin of such sporadic fatal cases of histoplasmosis appeared, at the time, to be inexplicable in the absence of any known source of the fungus. The present investigations, however, would indicate that the fungus is fairly widespread in the Transvaal. Although present knowledge only indicates its association with caves and with laboratories (Table I) it is more than probable that it also exists in other enclosed sites as described in the American literature. Evidence of this is to be found in the history of 3 miners who explored an old disused mine-shaft in the Klerksdorp district in 1950. Between 4 and 7 days later they developed a severe pyrexial illness which, because of the radiological features in a chest X-ray, was diagnosed as miliary tuberculosis. However they completely recovered after 3 weeks and have remained in good health since that time. It would appear in retrospect that they had contracted benign pulmonary histoplasmosis while exploring the old mine-shaft.

No doubt other similar acute pyrexial illnesses occur in South Africa which are, in the absence of general knowledge of the occurrence of histoplasmosis, erroneously diagnosed as influenza, virus pneumonia or some other similar condition. Such an erroneous diagnosis came to our attention in 1953, when a visiting overseas archeologist developed a pyrexial illness after exploring caves in Northern Rhodesia. This case was diagnosed, despite repeated negative blood-films, as malaria but a review of the history and a histoplasmin skin-test after his recovery strongly suggested benign pulmonary histoplasmosis. That the disease exists in Southern Rhodesia as well as in the Union, is further shown by the fact that a recent case of pneumonitis occurred in a government geologist after exploration of the Magwento caves in the Urugwe Reserve carried out with a view to determining their suitability for mining bat guano. But for the astuteness of his attendant physician this case might also have been diagnosed as virus pneumonia or some similar condition. Awareness of the existence of the condition in Southern Africa enabled his physician to establish the correct diagnosis.[34]

No history of association with caves or similar enclosed spaces is obtainable from the records of the 5 fatal cases to which reference has been made.[3,9,18] When the cases occurred it was not realized that it was important to elicit such information and it was not specifically asked for. It may be significant that one of the fatal cases was a miner[18] although it should be noted that in carrying out many hundreds of macroscopic and microscopic examinations of miners' lungs over the past 13 years one of us (JFM) has never observed any lesions resembling histoplasmosis. Since commencing this investigation a practice has been made, in cooperation with Dr. I. Webster of the Pneumoconiosis Unit, of searching for histoplasmosis and of carrying out special stains for the fungus in lung lesions in miners which might conceivably be due to that condition. To date no positive results have been obtained, but it is our intention to pursue this investigation by carrying out histoplasmin skin-tests on miners and on other groups of the population in different parts of the country with a view to determining the distribution of histoplasmosis. The only certain knowledge on this point in South Africa (apart from the clinical cases which have been reported and the information which forms the subject of this paper) is the demonstration by Jackson[35] that 0·7% of a group of 453 persons in the Cape Town area gave a positive histoplasmin skin-test, and by Dormer[10] that 0·0% of 893 Bantu in the Drakensberg area, 5·9% of 1,336 persons in the Durban area and 1·6% of 693 Bantu on the Natal coast gave positive histoplasmin skin-tests. One of us (H.I.L.), in 1949, showed an 8·0% positivity rate in European adults living in the Johannesburg area. As the majority of these subjects were drawn from laboratory staff this probably does not represent the true incidence in the general population and it is our intention to investigate other sections of the population so that a more accurate estimate of the incidence of the condition in the Transvaal may be obtained.

SUMMARY

The occurrence of benign pulmonary histoplasmosis in the Transvaal is described and evidence is presented to show that the 46 cases described in this paper derived their infection from the atmosphere of infected caves.

Our thanks are due to Prof. G. A. Elliott, Head of the Department of Medicine at the University of the Witwatersrand, for calling our attention to the original 3 cases which aroused our interest and for encouragement and advice during the investigation; to the Director of the Institute for numerous facilities; to Dr. H. H. Malherbe and Miss F. le Roux of the Virus Research Unit who, in their search for viruses, confirmed our findings by also isolating *H. capsulatum* from the lungs of animals exposed in infected caves; to Dr. M. L. Furcolow and Dr. C. Emmons for much helpful advice and cooperation; to Dr. M. Pringle for permission to mention the cases of the 3 miners; to many physicians for access to their cases and case records; to Mr. M. Ulrich

of the Institute's Photographic Department for Figs. 11-14, to Miss Tomkins of the Johannesburg General Hospital for Figs. 2-10 and to Miss J. Humphries for Fig. 1.

Our most grateful thanks are also due to the members of the Transvaal Spelaeological Society who, throughout the whole period of the investigations, have never flagged in their enthusiastic cooperation. In particular we would like to thank the 14 members of group 3 who voluntarily exposed themselves in an infected cave. We are also deeply indebted to all the other members of the Society who have so readily acceeded to our requests, both reasonable and unreasonable, for exposure of animals and other activities. We also wish to thank Dr. R. Kleinman and the members of the Cape Spelaeological Society who investigated histoplasmin skin-sensitivity rates for us in the Cape.

We are grateful to Eli Lilly and Co. for generous supplies of histoplasmin, to Prof. N. F. Conant for blastomycin, to Dr. R. W. Menges for haplosporangin, and to Dr. K. W. Walls for antigen for the complement-fixation test.

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THE OCCURRENCE OF A DENGUE-LIKE FEVER IN THE NORTH-EASTERN TRANSVAAL

I. CLINICAL FEATURES AND ISOLATION OF VIRUS

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Early in April 1956 our attention was drawn to the occurrence of an outbreak of a dengue-like fever in the North-Eastern Transvaal when one of us, F.P.R., was called by Dr. W. Chapman of Vereeniging to see in consultation one of 5 patients who had developed a feverish illness with unusual features soon after their return from a short fishing trip to this region.

CLINICAL FEATURES

This patient, Mr. V. aged about 30 years, with 4 companions had spent the previous (Easter) week-end Saturday, 31 March to Monday, 2 April in the neighbourhood of the Letaba river near the border of the Kruger National Park. On arriving back home in Vereeniging on the evening of Monday, 2 April, he felt pain in his left ankle as he got out of his car. That night he slept well.

On Tuesday, 3 April, his wrists became sore and the following day, Wednesday 4 April, one was visibly swollen. He now complained of pain all over the body particularly affecting the muscles and joints, and during the morning his throat became extremely sore. He was then given aureomycin. In the afternoon he vomited several times. He coughed a little, and almost continuously tried to clear his throat of accumulated mucus. The pain in his throat was too severe for him to be able to swallow. During the day he developed a severe temporal headache, and a non-itchy rash appeared.

He was seen that evening in consultation. On examination

it was noted that he was in much discomfort from severe sore-throat. He tried to swallow to get rid of the mucus, but choked in the attempt. On inspection his throat, particularly the soft palate and pharynx, was seen to be very oedematous. There was a diffuse reddening of the whole pharynx. The pharyngeal muscles moved badly and appeared almost paralysed, possibly owing to the severity of the pain. He was tender behind the angle of the jaw on the left side, where there was a slightly swollen tender lymphatic gland. Examination of the abdomen revealed no abnormality. Occasional rhonchi were heard in the chest, the heart sounds were closed, and the blood pressure was 120/80 mm. Hg. There was no neck rigidity. Kernig's sign was absent and the Babinski sign was flexor. The tendon reflexes were present and equal. He had a rash sparsely distributed over the front and back of the chest and the abdomen, and profusely in the lumbar region of the back and flanks. It was light on the forearms and did not involve the face or legs. There was slight palmar erythema. The rash was erythematous and consisted of macular spots varying in size from a pin-point up to 2 mm. in diameter. The maculae faded on pressure.

In the differential diagnosis tick-bite fever and other rickettsial diseases were considered, but they were excluded because the rash did not resemble that of a rickettsial disease. In was considered that the illness was probably caused by a virus. As it was at once clear that it was an unusual type of illness, arrangements were made for attempts to isolate and identify the causative agent.

The other 4 patients were not seen at this time, but were interviewed subsequently and each gave a history of having had a similar feverish illness.

Mr. J. developed pains in the right wrist-joint on 2 April. On 3 April his temperature was found to be 103°F and he had developed a macular rash and had a few petechiae on the palate. On 4 April the rash had disappeared but he complained of severe headache and pain in the back. Kernig's sign was positive. He was not delirious at any time. On 5 April a slight improvement was noted. On 6 April he was still feverish and noticed he could not focus properly. His temperature returned to normal on 8 April. In convalescence he has had several recurrences of painful stiffness of joints.

Mr. U first became ill on Monday evening, 2 April, the third day after his arrival on the borders of the National Park. He felt listless and 'washed out'. The following day he was feverish but his temperature was not recorded. His temperature on Thursday, 5 April, the 4th day of illness, was found to be 103°F. The following day it returned to normal.

During this feverish illness he developed pain in his right arm, which has persisted with periodic exacerbation since then. The pain affects the muscles attached to the outer side of the elbow, but these muscles are not tender. He now cannot pick up heavy weights and this disability is particularly noticeable in his work as an engineer concerned with heavy materials. He also occasionally suffers from pain across the back just below his ribs. These attacks are now becoming less frequent and less severe.

Soon afterwards our attention was called to several other cases of what appeared on the clinical report to be the same illness.

Dr. S. J. Fleishman, of Johannesburg, sent 2 patients of his, Mr. and Mrs. B, to us for an interview and the collection of blood specimens in convalescence.

Mr. and Mrs. B. had spent the period 29 March to 2 April at a farm on the Oliphants river. They stayed in fly-screened rondavels, which were not sprayed with insecticide during their visit, but they did not notice any mosquitoes. However, on Sunday evening they sat out on the stoep of the house and were bitten several times on the ankles. The bites itched for a few days afterwards. Milk was obtained from cows on the farm and there were a number of other cattle but they did not go near them and did not enter cowsheds.

Both Mr. and Mrs. B. became ill on Wednesday, 4 April, giving an incubation period of 3-6 days. Mr. B's illness began suddenly while on a business visit to Welkom in the Orange Free State. He had slight feelings of chilliness and developed a severe headache and marked congestion of the eyes. On 5 April his temperature at 9 a.m. was 104°F. He was treated with terramycin and remained in bed in his hotel at Welkom until Saturday, 7 April, when he got up and returned to Johannesburg. On Monday, 9 April he returned to work but felt weak. Soon after this he developed pains in the joints of the hands and the ankle joints. On 1 May he noticed pain in his knees and felt wobbly when he walked. He also had pain in the calf muscles and between the shoulder blades. At this time also he developed a nodule on the distal joint of the middle finger of his right hand. It was tender and somewhat itchy. These pains have persisted with some exacerbation up to the present.

Mrs. B. also became ill suddenly on the afternoon of Wednesday, 4 April. Feeling chilly she took her temperature and found it to be 101·5°F. Next morning, 5 April, her temperature was 102·2°F and she called in her doctor, who suggested she might have tick-bite fever and prescribed aureomycin. Later that day she developed pain in her elbows and back and slight headache, most marked behind her eyes. That evening her temperature was 103·3°F. She had no appetite and felt thirsty. On Friday, 6 April her temperature was 100·4°F. Her doctor prescribed a Daraprim tablet and another one for the following day. That evening her temperature returned to normal and she felt much better. The following day she felt shaky. On Monday, 9 April she returned to her work at a part-time job. She then noticed that she had no strength in her arm and could not lift things. She still had pain in the joints, especially in the distal and subdistal joints of the right thumb, which she could not bend. She also noticed a red rash on the front of her arms and on the outside of her right leg. The same week she developed pins-and-needles in the ring and middle fingers of her right hand. These still persist. During the day they are not so bad, but at night her whole arm is painful, with a burning feeling on the inside (flexor) surface and a cold feeling on the outside (extensor) surface. On Sunday, 29 April pain started in her ankle and in her right calf muscles and in her left leg and was particularly severe on getting up in the mornings. This painful stiffness wears off as she moves around. These symptoms still persist at the time of interview.

Another family of father, mother and 2 sons were on a visit at the same time to the farm where Mr. and Mrs. B presumably contracted their infection. All but one of the sons suffered from a similar illness.

Dr. I. Rudolph, of Middelburg, Transvaal, wrote on 4 April 1956 to say that 4 of a party of 3 adults and 8 children who had visited a farm bordering on the Kruger National Park had contracted an unusual illness. The party left Middelburg on Thursday 29 March 1956, spent 3 days on the farm and then returned home. On Tuesday 3 April, which was the 5th day after their arrival at the farm, the 3 of the party went down with severe headache, photophobia, aches and pains in the body, backache, and a mild sore-throat associated with fever. The temperature varied from 100-103°F. On examination these patients were found to have small red macules on the legs, which they thought were due to bites from small ticks, though none saw any ticks on themselves. One patient was found to have a palpable spleen and generalized adenitis with a rash somewhat resembling tick-bite fever. All these patients had taken Aralen, 1 tablet, before leaving for the Bushveld as a prophylactic measure against malaria.

Dr. T. W. Maré, of Phalaborwa (N.E. Transvaal), notified us of an outbreak of a similar illness which had affected a considerable number of the residents of the Phalaborwa area, in which mining operations had recently begun. The clinical picture of these cases was essentially similar to those described above. He reported

also that a considerable proportion of the Bantu employed on the mine had been affected.

Other cases of a similar illness in which the infection was presumably acquired on the borders of the Kruger National Park were also reported to us by the District Surgeon of Duivelskloof, Dr. L. J. Botha.

Comment

It was apparent that all these patients contracted their infection in an area bordering the Kruger National Park and in the neighbourhood of the Oliphants and Letaba rivers. Many of them became ill immediately after a visit to this region during the Easter week-end of 29 March to 2 April 1956. This was a long holiday week-end, when numbers of visitors came from the towns and cities of the highveld to the Eastern Transvaal lowveld. It was also the time of the year, at the end of the rainy season, when mosquitos and other insects were most prevalent.

From the accounts given by the individual patients, and the findings of the clinical examinations, a clear picture of this illness has emerged. The incubation period as deduced from the time and length of possible exposure to the time of onset is between 2 and 6 days. The onset was typically sudden, with acute pain in one or more joints, and feeling of chilliness. The temperature rose rapidly and was often over 103°F on the day after onset. The fever lasted 2-7 days, usually less than 5 days. During the febrile period some patients had a sore throat and headache, others did not complain of these symptoms. Suffusion of the conjunctiva, slight pain on movement of the eyes and photophobia were common. A macular erythematous rash involving the trunk and limbs developed on the 2nd-5th day of illness in many of the patients and was often followed by some desquamation of the skin soon after fading. The outstanding symptom was joint pain, which came on with dramatic suddenness and was often so severe as to prevent sleep, and which was aggravated by movement. Another characteristic feature of the cases has been a prolonged convalescence with recurrence of the joint pains, sometimes involving joints not previously affected. This painful stiffness of the joints is most marked in the mornings and is most painful at first movement, but on continuance of the movement the pain tends to regress.

Some patients developed nodules in relation to the joints of their fingers and others symptoms which suggested rheumatoid arthritis. However, the blood sedimentation was within normal limits. Other patients developed weakness of particular muscle-groups and sensory changes suggesting a localized neuritis. These symptoms of convalescence have persisted for several months, but gradually improvement has taken place and most patients have fully recovered their normal health within 6 months of the acute illness.

LABORATORY STUDY

As it was apparent that these patients were suffering from an unusual illness, arrangements were made to identify its cause.

Blood and throat swabs were taken from 3 of the Vereeniging cases in the acute phase of their illness. Dr. Rudolph collected blood also in the acute phase from 4 of his patients in Middelburg.

Blood was collected on several occasions from the Vereeniging cases in convalescence and from several other cases. A blood count made on blood collected on 4 April from Mr. U of Vereeniging gave the following result: Haemoglobin 17·6 g.%. Red cells 5·81 million per c.mm. White cells 12,700 per c.mm. (neutrophils 69%, monocytes 2·5%, lymphocytes 27·5%, eosinophils 1·0%, basophils *nil*). The red cells showed no pathological changes and malarial parasites were not detected in thick or thin blood-films.

The Widal and Brucella agglutination-tests gave negative results.

In the Weil-Felix test *Proteus OX* 2 gave a positive reaction in a dilution of 1 : 50 serum, a finding suggesting that tick-bite fever should be considered in the diagnosis. However, the specific rickettsial complement-fixation tests with *R. prowazeki, R. mooseri, R. conori var. pijperi, R. akari* and *R. burneti* yielded negative results, as did the virus complement-fixation tests with the viruses of herpes simplex, lymphocytic choriomeningitis and mumps.

The rickettsial and virus complement-fixation tests on the sera from Dr. Rudolph's patients from Middelburg also gave negative results.

The Paul-Bunnell tests also gave negative results.

Blood collected from 3 of the Vereeniging cases in the acute phase of the illness was also sent to the Laboratory for virus studies. On being received, the serum was separated from the clot and stored in a -20°C refrigerator for 2 weeks. The serum from each patient was then inoculated into 2 litters of baby mice in 0·015 c.c. amounts intracerebrally and in 0·03 c.c. amounts subcutaneously, and into 8 adult mice in 0·03 c.c. amounts intracerebrally. Each serum was also inoculated in 0·25 c.c. amounts into 2 tubes of monkey kidney-cell cultures. The adult mice inoculated with each of the 3 sera remained well. The baby mice inoculated with the sera of patients J and U respectively did not develop obvious signs of illness. Those inoculated with R's serum developed weakness and some became paralyzed, and all died within 1 week of inoculation.

On subinoculation of a suspension prepared from the brain and carcase of 2 of these mice into further litters of newborn mice, it was noted that most of them died on the 2nd day. These were harvested and a suspension prepared from their carcases.

Titration of Virus Suspension

The suspension prepared from infected mouse carcases was then titrated to determine the amount of virus present by the inoculation of tenfold dilutions each into a litter of 7 baby (one-day-old) mice. This test gave the following result:

Dilution of Virus Suspension	*Result of Test Survivors/Total*
10^{-1}	0/7
10^{-2}	0/7
10^{-3}	0/7
10^{-4}	0/7
10^{-5}	2/7
10^{-6}	6/7

In this experiment it was noted that most of the mice inoculated with the 10^{-1} and 10^{-2} dilutions lived longer than those inoculated with the 10^{-3} and 10^{-4} dilutions. On the second day after inoculation all the mice inoculated with the 10^{-4} dilution were dead, while all those inoculated with the 10^{-1} dilution, except one, were apparently normal. Presumably this greater virulence of the 10^{-4} dilution as compared with the 10^{-1} dilution is an example of interference by inactive or incomplete virus-particles in the more concentrated virus-suspension.

This virus suspension was preserved in a dry-ice cabinet and several protection tests were carried out with it.

Immunity Tests

A baby-mouse protection test was carried out with the sera from Mr. J. in the acute and the convalescent phase. In this test the sera were each mixed undiluted after inactivation at 56°C for 30 minutes with an equal volume of a 10^{-3} dilution of the R virus suspension. The mixtures were thoroughly shaken by hand for 3 minutes and then allowed to stand at room temperature for 2 hours, after which they were placed overnight in a +4°C refrigerator. Each mixture was then inoculated into 2 litters each of 7 one-day-old baby mice. In the same test were included litters of baby mice inoculated with a mixture of virus and Mr. B's convalescent serum and Mrs. B's convalescent serum respectively, as well as a negative control serum. The results of this test were as follows:

RESULTS OF BABY-MOUSE PROTECTION TESTS

Serum	*Result of Test Survivors/Total*	*Interpretation*
1. J acute phase	0/7	Negative
2. J acute phase	0/7	Negative
3. J convalescent phase ..	7/7	Positive
4. J convalescent phase ..	7/7	Positive
5. CM negative control ..	0/7	Negative
6. Mr. B. convalescent phase	7/7	Positive
7. Mrs. B convalescent phase invalidated by death of mother.		

It will be noted that the acute-phase serum gave no protection while the convalescent-phase serum fully protected against the R virus. It was thus concluded that this virus was the cause of J's illness and by implication the cause of the outbreak as a whole.

Although no acute-phase serum was available for comparative tests, it will be noted that Mr. B's convalescent serum also fully protected against the R virus.

A titration of the virus suspension showed that there were between 100 and 1,000 minimum lethal doses in the challenge dilution of 10^{-3} of R's virus.

Semliki Forest Virus Protection Test: We were informed by Dr. Smithburn that complement-fixation tests on the sera of several individuals convalescent from this illness showed some reaction with Semliki Forest virus. The protective value of an immune serum prepared in monkeys against Semliki Forest virus was then determined. In this test a 10^{-3} dilution of R virus was mixed with undiluted Semliki Forest virus monkey immune serum and, after shaking, the mixture as before was left at room temperature for 2 hours and then in a +4°C refrigerator overnight. Each mixture was then inoculated into a litter of 7 one-day-old baby mice. These were observed for 7 days. The results of this test are given in the following table:

Virus + Serum	*Result Survivors/Total*	*Interpretation*
R + CM negative control 1	0/7	Negative
R + CM negative control 2	0/7	Negative
R + SFAB 1	0/7	Negative
R + SFAB 2	0/7	Negative
R + J convalescent.. ..	7/7	Positive protection

SFAB = Semliki Forest immune serum

These results indicate that this virus is not Semliki Forest. Further tests are required to determine whether there is an immunological relationship demonstrable in neutralization tests.

Pathology in Baby Mice

Histological sections prepared from tissues of mice dying of the disease showed the most marked pathological changes in the brain. The neurones particularly of the basal ganglia showed degeneration with swelling of the nuclei and margination of the chromatin. Other cells showed pyknosis. In most mice there was little inflammatory infiltration, possibly because the time to death was so short. In some there was some perivascular cuffing with inflammatory cells. An acute inflammatory infiltration in the choroid plexus and an infiltration of neutrophil leucocytes at the base of the brain was observed in the sections from one mouse. The voluntary muscles showed eosinophil degeneration of small segments of some muscle fibres. Foci of necrosis associated with an acute inflammatory infiltrate were present occasionally in the adrenal glands. Collections of neutrophil leucocytes were seen sometimes in the spleen, the portal tracts of the liver, and the interstitial tissue of the lung.

Isolation of Virus in Tissue Culture

The tissue-culture tubes inoculated with the Vereeniging patients' sera were observed daily.

The tubes inoculated with U's serum did not show any marked changes during the 10-day period of observation. The tubes inoculated with R serum and J's serum on the 6th day showed some swelling of the peripheral cells. These changes may have been non-specific and were thought to be so at the time. There was no marked destruction of the cells.

The fluids were harvested on the 10th day and stored in a +4°C refrigerator for several weeks. Each was then inoculated into a litter of one-day-old mice. The mice inoculated with the fluid from U's cultures remained well and developed normally. The mice inoculated with the fluid from the tubes inoculated with R and J's sera developed signs similar to those of mice inoculated with R's virus and all were dead within 7 days. It seems certain that the virus had grown in these tissue-culture tubes inoculated with R and J's sera, but this was not proven. Sample mice from each of these litters were then handed over to Dr. C. Heymann, of Dr. Smithburn's team, as well as baby mice inoculated with the R virus, originally isolated by the inoculation of baby mice.

DISCUSSION

The illness suffered by these patients has such a similar picture that there is little doubt that all of them had the

same disease. The infection was contracted near the Western border of the Kruger National Park in the vicinity of the Oliphants and Letaba rivers; the association with this part of the bushveld may be of significance. In Phalaborwa the Africans apparently were affected equally with the Europeans, which suggests that they also had not previously had contact with the virus. This in turn implies either that the infection has been recently introduced or that it has a limited distribution, and that only those who enter this limited area are liable to contract it.

In its clinical picture the illness has many resemblances to dengue fever. The incubation period could be accurately assessed to vary from 2 to 6 days, probably most commonly 3 to 4 days. The onset was sudden with joint pain, followed by fever, headache, photophobia and general muscular pains associated with painful stiffness involving particular joints. The febrile attack lasted 3 to 6 days, but was followed characteristically by a prolonged convalescence during which the patients developed recrudescences of the painful stiffness of the joints and suffered from neuritic pains. In all these features there is a similarity to dengue fever. However, the virus isolated from one of the patients differed from the virus of dengue in several respects, notably in its ready transmission to baby mice, most of which died 2-3 days after inoculation. It was recalled that a dengue-like illness had been prevalent in the Newala district of the Southern Province of Tanganyika in 1952-53. This disease was given its Native name of Chikungunya fever, meaning 'that which bends up the joints'. The virus causing this epidemic was isolated and has been found to differ from the classical Hawaiian and New Guinea strains of dengue virus. It was considered possible that this virus and the one causing the outbreak in the Eastern Transvaal were similar.

A comparison was made with Chikungunya virus and other African arthropod-borne viruses, and the strain of virus isolated from the patient R, by Dr. K. C. Smithburn to whom this virus was handed for identification. Dr. Smithburn is head of a team, sponsored jointly by the Rockefeller Foundation, the South African Institute for Medical Research and the Poliomyelitis Research Foundation, engaged on a study of arthropod-borne virus diseases in Southern Africa. In the experience of its senior members, and in their facilities and equipment, this team was particularly well qualified to undertake the identification of this virus. At the same time as our study was in progress, this team was engaged independently in the investigation of other cases of the same infection. The results will be published separately, as will the details of their study of the immunological relationships of our virus, which showed that it is identical with or closely related to Chikungunya virus.

SUMMARY

In the autumn of 1956 several patients suffered from a dengue-like illness contracted whilst on a visit to the bushveld bordering on the Kruger National Park in the vicinity of the Oliphants and Letaba Rivers. This illness was characterized by an incubation period of 2-6 days, a sudden onset with joint pain, headache and muscular pain, and a maculo-erythematous rash which developed on the 2nd-6th day after onset. A striking feature was a prolonged convalescence during which recrudescences of painful stiffness recurred for several months. This illness also resembled Chikungunya fever originally described in Tanganyika in 1952-53.

A virus pathogenic for baby mice but not for adult mice was isolated from the serum of one of the patients. This virus was neutralized by the convalescent serum but not the acute-phase serum of another patient and therefore was considered to be the cause of the outbreak.

The study and identification of the virus has been undertaken by Dr. K. C. Smithburn and his findings will be reported separately.

We wish to thank those medical practitioners who recognized this illness as something unusual and asked for this investigation to be undertaken. In particular we would wish to thank Dr. W. A. H. Chapman, Dr. P. A. L. Vogts and Dr. H. Krige of Vereeniging, who first called our attention to patients suffering from the disease, and to Dr. I. Rudolph of Middelburg, Dr. S. J. Fleischman of Johannesburg, and Dr. T. W. Maré of Phalaborwa, who informed us of their patients' illness and arranged for collection of specimens from them.

We are also grateful to Dr. K. C. Smithburn, staff member of the Rockefeller Foundation, and his team for undertaking the study and identification of the virus concerned.

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FAMILIAL DISSEMINATED LUPUS ERYTHEMATOSUS

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The condition of subacute lupus erythematosus was first described in 1872 by Kaposi. The greatest advance came when Hargraves *et al.*[1] described the phenomenon of the L.E. cell, drawing attention to the specificity of the reaction. Originally considered to be a rare disease, more thorough investigations have shown that it is by no means an unusual disease-entity. In a recent review, Harvey *et al.*[2] have analysed 138 cases. In their series the disease has been commoner amongst females than males, and the age of onset has ranged from 2 to 67 years, although it occurred most commonly in the 2nd, 3rd and 4th decades. Originally described amongst white patients, it is being increasingly reported amongst negroes. Similar findings are reported by Ross and Wells[3] in their review of the literature.

Familial incidence has been reported by 5 previous authors who have described the occurrence of discoid lupus and disseminated lupus in siblings.[4–8] Shearn and Pirofsky[8] in a review of the literature found only 3 reports of a familial occurrence of disseminated lupus erythematosus.

Arthralgia and arthritis are well-known manifestations. In Harvey's series[2] of 138 cases, 90 complained of arthritis and/or arthralgia and 27 cases showed classical deformity of rheumatoid arthritis. In Ross and Wells' series[3] of 34 cases, 9 showed deformities of rheumatoid arthritis and 80% of their cases had complained of arthralgia.

The present cases are presented in order to draw attention to the occurrence of disseminated lupus erythematosus as a familial disease.

CASE 1

Mrs. L., a housewife aged 30 years, was originally seen in 1949, complaining of 'articular and non-articular rheumatism', present during the day, but more intensely so at night, and involving all limbs and back. She also complained of a sensation of pins and needles and an itchiness of hands and feet. The joint pains had been present for 3 years. During the first year she had been pregnant, and at this time she was 'treated with iodine for nerves'. On examination there was no evidence of swelling or joint crepitus, and no pain on movement. A blood count, Wassermann reaction and uric-acid estimation were normal, and a tentative diagnosis of 'allergic arthritis' was suggested. For this she was treated with anti-histaminics (Anthisan).

She was seen again 2 years later, when she stated that she was still subject to intermittent swellings of joints, especially the knee joints which were extremely painful. At this phase the patient felt well, and nothing of note was found on examination, but her doctor confirmed that during an attack she suffered 'crippling pain'. During the following 3 years she suffered periodic bouts of joint pain and swellings, and severe pain at other sites such as the substernal region and the soft tissues of the limbs, face and neck. She had lost weight.

On examination, the skin looked slightly dark for an otherwise fair person. Blood pressure was 150/95. There was no evidence of any rash and there was no hepatosplenomegaly or adenopathy. A clinical diagnosis of disseminated lupus erythematosus was suggested at this stage, and confirmed by the presence of L.E. cells in preparations of her blood.

In view of the chronicity, chloroquine and mepacrine were prescribed in preference to steroids. After a short time the patient reported that she was unable to tolerate the chloroquine, but the mepacrine (0·1 g. t.d.s.) produced relief. A point of interest was the fact that a tot of champagne would precipitate an attack of severe joint-pains which passed off after a few days. For a short while she discontinued treatment owing to skin discoloration, but resumed when the pains returned. Four months after commencement of treatment the mepacrine was replaced by chloroquine (the dose of which varied from 1 tablet t.d.s. to 1 tablet a day, depending upon the absence of symptoms), and since then improvement has been striking and lasting. Her weight has increased, she looks well, and she has a much more optimistic outlook on life now that she is free from the excruciating pain. Periodically, some pain occurs in joints, in the chest, etc., but this is soon controlled by temporary increase in the dose of chloroquine.

Laboratory Investigations

A full *blood-count* gave the following results:

The haemoglobin has remained at a normal level of 14-15 g. per 100 ml. Leucocytes have fluctuated between 4,000 and 8,000 per c.mm., with a relative lymphocytosis—polymorphs 40%, lymphocytes 57%, monocytes 2%, eosinophils 1%. Sedimentation rate (Wintrobe) 27 mm. in 1 hour. Shortly after the diagnosis had been made and treatment had been commenced, there was a leucocytosis of 14,000 per c.mm., with a normal differential count. The blood smear showed the presence of anisocytosis with target cells. The most recent count (during remission) was as follows—haemoglobin 15 g. per 100 ml., P.C.V. 45%, M.C.V. 87 cμ, M.C.H.C. 33%, leucocytes 9,600 per c.mm. (neutrophils 67%, monocytes 1%, lymphocytes 30%, eosinophils 2%), sedimentation rate (Wintrobe) 6 mm. in 1 hour; the platelets were normal in numbers and in appearance.

L.E. cells and the L.E. phenomenon have been demonstrated on several occasions and are still present in moderate numbers in spite of the patient's clinical remission. These have been demonstrated on smears from clotted blood and, in large numbers, on smears prepared from the buffy-coat layer of defibrinated blood which has been incubated at 37° C for 2½ hours.[9]

Uric acid 3·5 mg. per 100 ml., Wassermann, V.D.R.L. and Ide tests negative.

Urine albumen present (+). Microscopic examination of urine showed the presence of 10-15 erythrocytes and polymorphs per high-power field, and numerous epithelial cells.

Blood urea 31 mg. per 100 ml., thymol turbidity test 3·5 units, thymol flocculation test one plus, colloidal-red test four plus, cephalin-cholesterol flocculation test (24 hour reading) negative, Takata-Ara reaction negative, zinc-sulphate turbidity 11·2 units, total lipid 446 mg. per 100 ml., alkaline phosphatase (King-Armstrong) 8·6 units, total bilirubin 0·8 mg. per 100 ml., cholinesterase 97% of average normal activity, mucoprotein 137 mg. per 100 ml.

Before treatment, total protein was 6·7 g. per 100 ml., with albumin 3·4 g. and globulin 3·3 g. The most recent analysis, using the Antweiler micro-electrophoretic apparatus, during a period of remission showed a total protein of 7·4 g. per 100 ml.,

with albumin 45·7%, alpha-1 globulin 4·3%, alpha-2 globulin 7·7%, beta globulin 12·9%, gamma-1 globulin 3·9%, gamma-2 globulin 25·5%.

CASE 2

Mrs. A., aged 45 years, is a sister of the first patient, Mrs. L. The patient was first seen in May 1956, with a history of rheumatoid arthritis of 6 years' duration. The story and clinical picture was typical of rheumatoid arthritis with a marked degree of

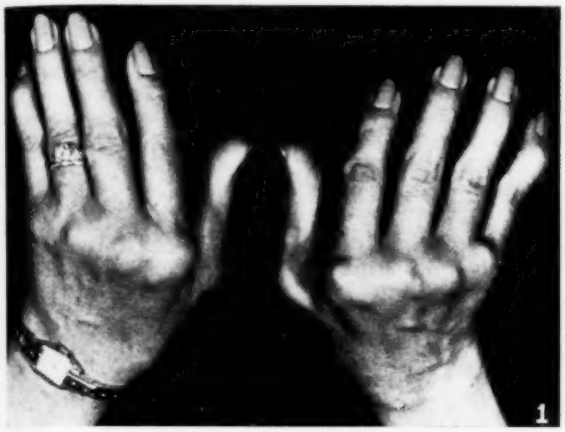

Fig. 1. Case 2. Showing changes in both hands characteristic of rheumatoid arthritis. Note degree of ulnar deviation.

ulnar deviation of both hands (Fig. 1). For 3 years she had been taking cortisone, but discontinued this about 1 year ago, when she found that aspirins were giving as much benefit.

During the past 6 months she had lost 21 lb. in weight, and the pain and stiffness of the shoulder joints have been increasing. She was hospitalized a few years ago for an attack of 'pericarditis'.

Examination revealed a poor state of nutrition. Weight 90 lb., pulse rate 100 per minute, with extrasystoles. The heart was not enlarged. Other systems showed no abnormalities. There was no hepatosplenomegaly and no skin lesions on the face or elsewhere.

In the normal course of events, the diagnosis of rheumatoid arthritis would have been accepted, but in view of the diagnosis of lupus erythematosus in her sister, the patient's blood was examined for L.E. cells. These were found in abundant numbers (see below).

With the establishment of a diagnosis of L.E., the patient was treated with chloroquine, with a satisfactory response, as evidenced by increase in weight, disappearance of chest pains, relief of joint pains and general improvement in appearance.

Laboratory investigations

A full blood-count gave the following results:

Haemoglobin 15 g. per 100 ml., M.C.V. 87 c.μ, M.C.H.C. 35%, P.C.V. 43%, leucocytes 4,700 per c.mm., with polymorphs. 52%, lymphocytes 43%, monocytes 2%, eosinophils 3%; platelets were normal in numbers and appearance; sedimentation rate (Wintrobe) 15 mm. in 1 hour; reticulocytes 1·3%. The smear showed the presence of anisocytosis with a number of target cells. L.E. cells were demonstrated in preparations from clotted blood, and in abundant numbers from the buffy coat of defibrinated blood, which was incubated at 37°C for 2½ hours. The L.E. cells were characteristic, and clearly distinguishable from tart cells and nucleophagocytes (Fig. 2). Rose's sheep-cell agglutination test was negative on one occasion and positive 10 days later. Wassermann, Kahn and Ide tests were negative. Uric acid 3·8 mg. per 100 ml., urea 17 mg. per 100 ml., and urobilin one plus. The direct Coombes test was positive. Thymol turbidity test 8·0 units, thymol flocculation test four plus, colloidal-red test four plus, cephalin-cholesterol flocculation test (24-hour reading) four plus, Takata-Ara reaction three plus, zinc-sulphate

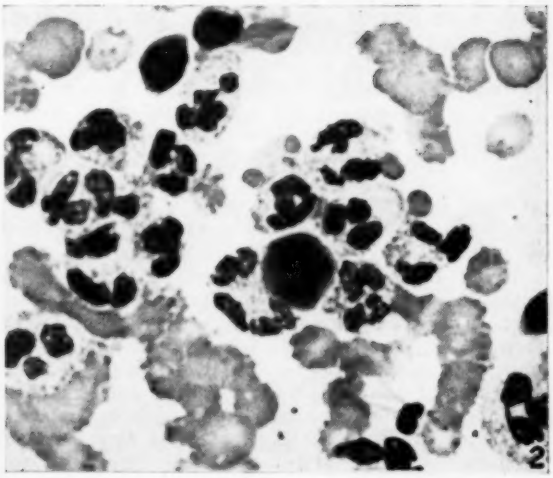

Fig. 2. Case 2. Smear of the buffy coat of defibrinated blood, showing L.E. cells. Wright's stain × 900.

turbidity 46·0 units, total lipid 511 mg. per 100 ml., alkaline phosphatase (King-Armstrong) 10·3 units, total bilirubin 0·6 mg. per 100 ml., cholinesterase 69% of average normal activity, mucoprotein 112 mg. per 100 ml., total cholesterol 156 mg. per 100 ml., percentage esters to total cholesterol 73%. An electrophoretic protein analysis (Antweiler) gave the following results—total protein 8·7 g. per 100 ml. (albumin 28·9%, alpha-1 globulin 3·6%, alpha-2 globulin 9·5%, beta globulin 13·4%, gamma-1 globulin 5·4%, gamma-2 globulin 39·2%).

Urine. Albumen absent; no significant urinary microscopical findings.

X-ray of chest (by Dr. C. Komins) showed pleural thickening in both costo-phrenic angles, associated with increased broncho-vascular markings and slight unfolding of the ascending arch of the aorta. The bilateral pleural changes associated with the increased broncho-vascular markings have been described as fairly typical of disseminated lupus erythematosus.[10–11]

DISCUSSION

Most workers agree that L.E. cells are specific in cases of lupus erythematosus and that previously reported cases of the L.E. cells occurring in other diseases have been due to failure to distinguish the true L.E. cells from tart cells and from nucleophagocytes. These latter cells occur in hypersensitive states and in a miscellany of diseases. The true L.E. cells show a smoky homogenous appearance, with a tendency to rosette formation and the absence of a dark outer rim in the inclusion body. Nucleophagocytes may occur in rheumatoid arthritis, but true L.E. cells are not found.[12]

Recent work has shown that the development of the L.E. cells requires activation by some substance, probably derived from platelets, which reacts with susceptible cells, and that previously injured cells are most susceptible. Allowing blood to clot, and defibrinating of blood both cause this degree of injury to the red cells.[13–15] The technique of using defibrinated blood has been preferred, as the leucocytes undergo less degeneration and a more satisfactory smear is produced.

Approximately 70% of cases show a moderate to severe normochromic normocytic anaemia usually associated with a leucopenia. In a small percentage of

cases there may be a leucocytosis. The sedimentation rate is increased in a very high proportion of cases and a thrombocytopenia is sometimes evident. The anaemia is frequently related to the duration and severity of the illness, and is usually due to retarded erythropoiesis. The blood smear often shows minor abnormalities of anisocytosis, basophilic stippling and Howell-Jolly bodies. The cases described above showed the feature of target cells, without any anaemia. These cells occur frequently in various forms of liver disease and appear here to correlate with the 'liver function tests'.

Acute disseminated lupus erythematosus can be regarded as an acute systemic disease of the collagen type. The incidence of females suffering from it varies from 68·7% to 95·7% and it is most commonly observed during the child-bearing age.[8] Diagnosis of this condition may become extremely difficult where skin lesions are absent. Acute disseminated lupus erythematosus may effect any organ of the body and the diagnosis must be seriously considered in those cases where skin lesions are absent, if some or several of the following signs, symptoms or abnormal laboratory results are evident: Arthralgia or arthritis, signs and symptoms of pericarditis and/or pneumonitis, or an unexplained, intermittent temperature. Positive serological tests for syphilis are found in a small percentage of cases. Protein electrophoretic analysis of serum, in a high percentage of cases, show a lowered albumin, a high alpha-2 globulin and a high gamma globulin. The commonly used flocculation and turbidity tests, e.g. thymol turbidity, thymol flocculation and the cephalin-cholesterol flocculation tests are usually positive, mainly because of abnormal serum-protein fractions, though evidence of hepatic dysfunction, as shown by excessive bromsulphalein retention, may be found in a small percentage of cases. Renal manifestations, as evidenced by proteinuria, haematuria and/or casts are prominent in over 50% of cases. In 20% of cases the blood-urea level is elevated.

Anaemia was not present in case No. 1. When L.E. was diagnosed, a leucocytosis was present. A leucopenia was subsequently evident on occasions. The sedimentation rate was elevated and the albumin globulin ratio was approximately 1 : 1. Serological tests for syphilis were negative. The thymol turbidity and flocculation tests were abnormal, mainly owing to alterations in the serum-globulin fraction. Although the blood-urea level was not elevated, urine examination showed proteinuria and haematuria.

In case No. 2 there was no anaemia, the leucocyte count was at the lower limits of normal, and the sedimentation rate was only slightly elevated. Serological tests for syphilis were negative. The blood urea was within normal limits and neither protein nor any significant microscopical changes were found on examination of a specimen of urine. The patient showed a marked disturbance of protein, namely a lowered albumin, a considerable rise in the gamma-globulin fraction and a slight increase in the alpha-2 fraction. The turbidity as well as flocculation tests were markedly disturbed. Bromsulphalein tests were not carried out in either case. The cholinesterase in case 2 was on the low side, but normal in case 1. In both cases there was an elevated serum-mucoprotein level.

SUMMARY

Two sisters, one presenting with severe arthralgia and transient joint swellings, and the other as a classical case of chronic rheumatoid arthritis were found to be suffering from acute disseminated lupus erythematosus. The relative literature has been briefly reviewed, and laboratory tests carried out on these cases are described and discussed.

Our thanks are due to Mr. Ulrich of the South African Institute for Medical Research for the photographs used in this paper.

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THE MANAGEMENT OF BROW PRESENTATION

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In 1952 a series of 21 cases of brow presentation was published,[1] occurring in 18,737 deliveries in the maternity institutions attached to the University of Cape Town. This represented an incidence of 1 : 892. Attention was then called to the controversy in the literature in regard to the incidence, prognosis and treatment of this condition. The incidence is variously placed between 1 : 600 (Queen Charlotte Text-book of Midwifery[2]) and 1 : 4,000 (Munro Kerr and Chassar Moir[3]). In the present series of cases, which have been collected during the 4 years ended 30

June 1956 in the obstetrical unit of the University of Cape Town, 57 cases of brow presentation occurred in 30,106 deliveries. This is an incidence of 1 : 528 cases. If the series published in 1952 is incorporated, there were 78 brow presentations in 48,843 deliveries, an incidence of 1 : 626 cases.

Prognosis

1. *Spontaneous Delivery.* Wilfred Shaw[4] stated that spontaneous delivery was one of the rarest occurrences with brow presentation and that it must not be anticipated. Chassar Moir[3] stated that it occurred only in 4-6% of cases. Fink[5] and Ingerslev,[6] in large published series, claim that spontaneous delivery occurred in about 40% of cases. In the present series spontaneous delivery occurred on 7 occasions (an incidence of 12·3%). In the 1952 series it occurred only once (4·76%). Thus in 78 cases of brow presentation in the 2 series 8 patients were delivered spontaneously, this being an incidence of 10·3%. The figure could no doubt be raised by delaying interference in the second stage.

2. *The Mother.* Stander gives the maternal mortality in brow presentation as 3-5%.[7] Many authors have reported a worse prognosis.[1] Weiss,[8] however, described 29 cases without the loss of a mother or child. There was 1 maternal death in the present series, an incidence of 1·75%. Combined with the 1952 series, in which no mother died, the incidence was 1 : 78 cases or 1·28%. The patient died of eclampsia. She had 4 fits before delivery. Internal version was performed after an attempted forceps delivery had failed. The abnormal presentation may well have been a contributory cause of this death. Surprisingly, eclampsia occurred in 3 of the 57 cases of brow presentation.

There were no cases of rupture of the uterus in the present series and, as there were 2 cases in the 1952 series, the incidence is 2 in 78 or 2·56%.

3. *The Child.* In the first series there were 7 stillbirths and no neonatal deaths in 21 cases of brow presentation, which represents a foetal loss of 33%. In the second series there were 7 stillbirths and 3 neonatal deaths in 57 cases, which is a foetal loss of 17·5%. Among the latter were 2 cases of gross foetal abnormality, and 2 in which the foetal heart was not heard on admission. Therefore with institutional treatment the corrected foetal loss is 11·3%. Foetal mortality ranging from nil to 50% has been recorded in the literature.[8,9,10]

Diagnosis

Abdominal palpatation may suggest the abnormal presentation, but the diagnosis is almost always made or confirmed by vaginal examination. Occasionally, late in labour, a large caput may obscure the landmarks. In doubtful cases a diagnosis must always be made, either with the aid of X-ray examination or by vaginal examination under anaesthesia. If this rule is observed, neglected cases and their severe consequences will not occur.

Treatment

The controversy in regard to treatment has been discussed.[1] Gibberd[11] holds that interference should take place early, as soon as the diagnosis has been made. He advises that when the position has been corrected the membranes should be ruptured and a tight binder applied. This, however, may decrease the foetal oxygen supply and increase the risk of intra-uterine sepsis in a prolonged labour. An anaesthetic may be necessary, and yet there is no guarantee that the new position will be maintained. Sjovall,[12] and Munro Kerr and Chassar Moir,[3] correct the presentation only when the membranes have ruptured, irrespective of the dilatation of the cervix. This will require an anaesthetic, when delivery may not be effected for a number of hours, and the life of the foetus may thus be jeopardized. If the head is mobile the corrected presentation is unlikely to be maintained. In 17·5% of Ingerslev's cases there was difficulty at the brim, which caused 66% of the stillbirths. De Lee and Greenhill[13] advise intelligent expectancy, and correction of the presentation should it persist after an hour in the second stage; and it is this method we favour. As long as the maternal and foetal conditions remain good and labour is progressing, it is not deemed necessary to interfere. Should there be an added complication, or should a complication arise during labour, it may be treated according to indications, or one may resort to Caesarean section.

The mode of delivery in the present series and the 1952 series is shown in Table I. Although the stillbirth rate has

TABLE I. MODE OF DELIVERY IN 78 CASES OF BROW PRESENTATION

	1st series (1952)	*2nd series* (1956)	*Total*
Spontaneous delivery	1 (4·8%)	7 (12·3%)	8 (10·3%)
Conversion to face: spontaneous delivery.	1 (4·8%)	3 (5·3%)	4 (5·1%)
Conversion to face: forceps delivery.	3 (14·3%)	9 (15·8%)	12 (15·4%)
Conversion to vertex: forceps delivery.	1 (4·8%)	9 (15·8%)	10 (12·8%)
Forceps only	0	2 (3·5%)	2 (2·6%)
Internal version	4 (19·0%)	6 (10·5%)	10 (12·8%)
Caesarean section	7 (33·3%)	20 (35·1%)	27 (34·6%)
Craniotomy	2 (9·5%)	1 (1·8%)	3 (3·8%)
Laparotomy (ruptured uterus).	1 (4·8%)	0	1 (1·3%)
Willett's forceps	1 (4·8%)	0	1 (1·3%)
Total	21	57	78

been reduced from 33% in the first series to 17·5% in the second, there is yet room for improvement. Of the 10 babies lost in the second series, as stated above 2 were due to con-

TABLE II. STILLBIRTHS AND NEONATAL DEATHS IN RELATION TO MODE OF DELIVERY

Mode of Delivery	*No. of cases*	*Stillbirths*	*Neonatal death*	*Remarks re dead baby*
Spontaneous delivery: as face	4	1	0	No foetal heart-sounds on admission
as vertex	2	0	0	
as brow	1	1	0	Large congenital goitre
Conversion to face: spontaneous delivery	3	0	1	4 lb. 15 oz.
Conversion to face: forceps delivery	9	0	0	
Conversion to vertex: forceps delivery	9	0	1	Foetal ascites
Forceps only	2	0	0	
Internal version	6	3	1	3 lb. 4 oz. (neonatal death)
Caesarean section	20	1	0	1st twin.

genital abnormalities and in 2 other cases the foetal heart was not heard on admission. These deaths could not have been prevented; but when they are excluded the stillbirth rate is still 6 in 53 cases or 11·3%. Of these 6 deaths, 4 followed internal version (Table II). In the 2 series combined, internal version was performed on 10 occasions with the loss of 5 babies, a foetal mortality of 50%. The opinion is therefore held that, with modern facilities, internal version has little or no place in the treatment of brow presentation. In cases where the foetus is alive, internal version results in far too high a foetal mortality. In neglected cases in which the foetus has succumbed, the procedure is not safe enough for the mother. The temptation to perform internal version arises in the labour ward when other methods of vaginal delivery have failed. It is a temptation which all engaged in obstetrical practice have suffered. To avoid it, vaginal manipulations should be performed in a fully prepared theatre and immediate Caesarean section should be resorted to if other safe methods of delivery fail.

SUMMARY

A series of 57 cases of brow presentation which occurred in the obstetrical unit of the University of Cape Town in the past 4 years is presented. The foetal mortality was 17·5% as compared with 33% in a series of 21 cases published in 1952.

The incidence, prognosis and management of cases is discussed. The methods of delivery adopted in the 2 series are shown. It is suggested that if the foetal mortality is to be further reduced, internal version as a form of treatment should be rejected, unless it is done in a theatre fully prepared for immediate Caesarean section by an obstetrician experienced in this art.

I wish to thank Prof. James T. Louw for allowing this survey to be conducted, and for his interest and advice; the Superintendents of the Peninsula Maternity, Somerset, Mowbray and St. Monica's hospitals for allowing me access to the records; and Dr. David Friedlander for his advice and criticism.

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THIRD ANNUAL REPORT (1955) OF THE DEPARTMENT OF ANAESTHESIA, GROOTE SCHUUR HOSPITAL, CAPE TOWN, AND UNIVERSITY OF CAPE TOWN

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Staff

Two temporary posts of anaesthetist were added to the strength of the Department. The number of anaesthetics administered in 1955 was 10·3% greater than in 1954.

Equipment and Drugs

It continues to be difficult to ensure regular and expert care and servicing of anaesthetic machines and oxygen therapy equipment. This factor, coupled with pressure of other work, has forced the Department to abandon its attempts to provide an oxygen therapy service.

Anaesthetic record cards are completed in respect of all patients receiving an anaesthetic. No machinery is available for statistical analysis of this vast volume of information.

The cost of administering an anaesthetic will be the subject of a separate report.

Teaching

Continued efforts are directed to the improvement of undergraduate and postgraduate teaching. A second refresher course in Anaesthesia intended for general practitioners, was held from 10 to 14 January, and the winter seminar course was again held. At the invitation of other Departments, several lectures were delivered on the place of Anaesthesia in special subjects, for example in Obstetrics, or of special subjects in Anaesthesia, for example, Anatomy. This feature of undergraduate training contributes to better integration.

An examination for the degree of Master of Medicine in Anaesthesia was held in November; 3 candidates passed.

Research

Pressure of routine duties has severely interfered with research activities but these continue on a reduced scale. Studies of arterial oxygen saturation during anaesthesia, on carbon-dioxide concentrations in anaesthetic apparatus, and on respiratory minute-volume changes during anaesthesia, were undertaken. Ballisto-cardiographic studies of cardiac output during anaesthesia, and of ECG and EEG changes during anaesthesia, are under way. A two-channel recording unit is under construction in the Department and will facilitate these studies.

Anaesthetics Administered

The volume of work done during the year is indicated in Tables I, II and III, where it is compared with the figures for previous years.

The total of 16,625 anaesthetics covers the work done in the teaching hospitals which we serve—an increase of 10·3% over the figure for 1954. With the addition of 1,746 dental anaesthetics the grand total becomes 18,371, an over-all increase of 7·1% on 1954.

Numbers are not, of course, the only reflection of the work done and Table III shows the numbers divided into two groups. In the first group are entered those anaesthetics for major surgery which usually last longer than 1 hour, while in the second group are those anaesthetics which ordinarily do not occupy more than 1 hour and usually are much shorter. This arrangement

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TABLE I. TOTAL NUMBER OF ANAESTHETICS ADMINISTERED FOR SURGICAL, OBSTETRICAL AND MEDICAL PURPOSES (DENTAL ANAESTHETICS ARE EXCLUDED)

Year	Total Anaesthetics	% Increase per annum
1945	6,710	—
1950	10,724	—
1951	11,891	10·8
1952	13,216	11·1
1953	14,966	13·2
1954	15,076	0·7
1955	16,625	10·3

TABLE II. SHOWING ANAESTHETICS ADMINISTERED FOR VARIOUS SURGICAL SERVICES

Theatre	1953	1954	1955
General Surgery	4,499	4,449	4,559
Gynaecology	2,622	2,487	3,849
Obstetrical Service	—	292	379
E.N.T.	2,573	2,348	2,373
Casualty Surgery	1,943	2,035	1,836
Orthopaedics	1,621	1,598	1,731
Urology	674	606	594
Ophthalmology	455	506	537
Neurosurgery	411	593	668
Medical, etc.	168	162	99
Totals	14,966	15,076	16,625
Dentals	1,179	2,079	1,746
Grand Totals	16,145	17,155	18,371

TABLE III. ANAESTHETICS ADMINISTERED FOR VARIOUS TYPES OF SURGERY

Major Anaesthetics	1953	1954	1955
Gastric resection	277	255	288
Biliary system	166	186	232
Large bowel and rectum	29	76	134
Cardiac	39	69	44
Other intrathoracic	81	66	97
Major jaw and tongue	14	31	36
Oesophagectomy	17	14	22
Intracranial	222	233	255
Other neurosurgical	189	360	413
Open bone operations	566	715	790
Mastoidectomy	91	116	178
Fenestration	5	4	5
Nephrectomy	42	34	36
Transabdominal urological	191	148	167
Vesicovaginal fistula	28	38	46
Hysterectomy	318	336	429
Pelvic exenteration	—	28	43
Laryngectomy	—	—	7
Adrenalectomy	—	—	10
Total	2,275	2,709	3,232
Minor Anaesthetics			
Plastic surgery	428	438	483
Sympathectomy	17	70	70
General Surgery	3,431	3,244	3,150
Manipulations	1,055	883	941
Nasal sinuses	103	55	75
Tonsillectomy, etc.	2,374	2,173	2,108
Ophthalmic	455	506	537
Bronchograms	72	75	49
Medical	96	87	50
Casualty surgery	1,943	2,035	1,836
Transurethral	97	87	79
Other urological	344	337	312
Colporrhaphy	146	152	168
Caesarian section	237	277	241
Obstetrical	—	119	134
Other gynaecological	1,893	1,829	3,163
Total	12,691	12,367	13,393

shows that during 1955 there was an increase of 19·3% over 1954 in the larger and longer operations while there was an increase of only 8·1% in the smaller and shorter operations. Considering the time factor alone, there was thus an increase of 27·4% on the time spent in administering anaesthetics in 1954. If numerical and time factors are combined, the over-all increase in work done is of the order of 37%. This calculation does not take into account time spent on subsidiary services, including pre-anaesthetic examination and post-anaesthetic follow-up.

This very large increase in the work achieved by the Department is due in part to the 15% increase in full-time staff and in part to constant efforts to improve the efficiency of the Department. In this respect we were aided considerably by the cooperative efforts of members of the Surgical and Gynaecological Divisions.

Deaths Associated with Anaesthesia

In Table IV are listed those deaths which fall within the scope of Section 86 of the Medical, Dental and Pharmacy Act No. 13 of 1928. There were a total of 23 such deaths during the year, of which 6 occurred during the operative period. The remainder occurred at varying times during the immediate post-anaesthetic

TABLE IV. DEATHS FALLING WITHIN THE SCOPE OF SECTION 86 OF ACT NO. 13 OF 1928

Age	Operation
	Died in the Operating Theatre
Adult	Craniotomy
Child	Subdural haemorrhage
Adult	Craniotomy
Child	Mediastinal tumour
Adult	Oesophagectomy
Adult	Peritonitis—perforated small bowel
	Died Post-operatively
Adult	Cranio-angiogram
Adult	Cranio-angiogram
Adult	Strangulated vantral hernia
Adult	Mitral valvotomy
Adult	Ovarian cystectomy
Adult	Radical resection of parotid
Adult	Craniotomy
Adult	Peritonitis—large bowel perforation
Adult	Resuture burst abdomen
Adult	Subdural haematoma
Newborn	Congenital diaphragmatic hernia
Adult	Glossectomy
Adult	Depressed fracture of skull
Adult	Ruptured spleen
Adult	Peritonitis—perforation of large bowel
Newborn	Peritonitis—congenital atresia of bowel with perforation
Adult	Re-exploration of common bile-duct.

TABLE V. CLASSIFICATION OF DEATHS LISTED IN TABLE IV

Anaesthetic as	*Operative*	*Post-operative*
1. The Major Factor in the Death	3	2
2. A Necessarily Contributing Factor, i.e. plus existing disease or plus surgical trauma, etc.	3	10
3. An Unnecessarily Contributing Factor (see body of report)	—	4
4. Non-contributary, i.e. surgical death, nursing error, etc.	—	1
Totals	6	17

period up to 24 hours after the termination of the anaesthetic. Some of these deaths are still under magisterial review and details are accordingly omitted, but in Table V we give a tentative classification. Of the 4 classes in this table, the first lists the anaesthetic as the major cause of death. It is here necessary to emphasize that to say that the anaesthetic is considered the major cause of death does not mean that the death was necessarily preventable.

The second class includes those instances where the anaesthetic was necessarily a contributory factor but not a major factor. As examples we quote those patients whose pre-existing disease

measurably increased the risk of surgery and anaesthesia or those patients in whom surgical trauma added a greater burden than usual to the stress of the operation.

In the third class are those deaths where anaesthesia contributed unnecessarily: that is to say, the anaesthetic appeared to have contributed to the death but would not ordinarily be expected to do so under similar circumstances. Here are included all cases of uncertain classification by virtue of the fact that uncertainty exists.

In the final class are listed those instances where death was frankly due to causes other than the anaesthetic. Here would be listed deaths due to surgical error or accidents beyond the control of the anaesthetist.

It will be seen that in only 9 patients was the anaesthetic a major factor or an unnecessarily contributing factor. This gives an incidence of 1 death per 2,041 anaesthetics, a rate which compares favourably with similar mortality rates elsewhere.

We are indebted to Dr. N. H. G. Cloete, Medical Superintendent of the Groote Schuur Hospital, and to the Dean of the Faculty of Medicine of the University of Cape Town, for permission to publish this report.

NATIONAL CANCER ASSOCIATION OF SOUTH AFRICA

NEW QUARTERLY BULLETIN

The first issue of the *South African Cancer Bulletin* (January-March 1957), an official publication of the National Cancer Association of South Africa, has just been received. It is edited by Dr. H. A. Shapiro, published by Messrs. Juta and Co. Ltd., Cape Town, and printed by Cape Times Ltd. The bulletin is a well produced journal which is a credit to the editor, the publisher and the printer.

This issue contains two main articles, viz. one on 'Psychiatric Aspects of Malignant Disease' by Dr. C. H. Hardin Branch, of the University of Utah, Salt Lake City, Utah, and one on 'Gastric Carcinoma' by Dr. Cyrus E. Rubin, of the University of Washington, Seattle, Washington; as well as a case report entitled 'Malignancy in Fibroids', by Dr. J. Gluckman, Johannesburg, the first of a series 'Pathological Case Book'.

A prominent feature is constituted by 11 pages of diagrams printed in two colours illustrating the stages of carcinoma of the cervix and the lymphatic spread of cancer in the female genital tract. Abstracts are given of 14 articles. An In Memoriam notice is included concerning the recent death of Mr. Hugh Solomon, who for over 20 years devoted himself unsparingly to the work of the National Cancer Association.

Messages of welcome to the new Journal are contributed by the Minister of Health (Hon. J. H. Viljoen), Prof. Sir Charles Dodds, Chairman of the Scientific Advisory Committee of the British Empire Cancer Campaign. Dr. Charles S. Cameron, Editor of *Ca*, the Bulletin of the American Cancer Society, and Dr. Lewis S. Robertson, President of the National Cancer Association of South Africa.

The Medical Association of South Africa and the *South African Medical Journal* welcome the appearance of this publication of the National Cancer Association and wish the National Association success both with their journal and the important part they have undertaken in the campaign against malignant disease in this country.

SOUTH AFRICAN MEDICAL CONGRESS, DURBAN, SEPTEMBER 1957

The forty-first Congress and the twentieth Scientific Meeting of the Medical Association of South Africa will be held in Durban, Natal, during the week, Monday, 16 September to Saturday, 21 September 1957.

The Headquarters of Congress will be at Red Cross House, Old Fort Road, Durban.

OPENING CEREMONY

The Opening Ceremony will take place on Monday, 16 September at 8.30 p.m. in the City Hall, Durban, and Congress will be officially opened by our Patron, His Excellency the Governor-General, Dr. The Honourable E. G. Jansen. It is customary to wear academic dress at the Opening Ceremony and those members who wish to hire graduate gowns and hoods for Congress should make arrangements with Messrs. Woolfsons, Chester House, 388 West Street, Durban, before 31 July 1957.

TRAVELLING FACILITIES AND ACCOMMODATION

Members wishing to make hotel reservations for Congress week are requested to communicate with their nearest South African Railways' Travel Bureau or Station Master. This approach will prevent double booking, save correspondence and ensure satisfactory travelling arrangements.

THE SCIENTIFIC EXHIBITION

The Scientific Exhibition will be held in the Drill Hall, Durban, on the opposite side of Old Fort Road to Red Cross House and the Trades Exhibition. This exhibition will be one of the main features of the 1957 Congress and offers a unique opportunity for the demonstration of scientific work carried out in the Union, not only by officially sponsored research units, but by individual medical practitioners, who will play a big part in the exhibition. Members of the Association who wish to take part in this exhibition should write to the Convener of the Scientific Exhibition Committee, Mr. B. W. Franklin Bishop, 711 Payne's Building, West Street, Durban, as soon as possible.

THE TRADES EXHIBITION

The Trades Exhibition will be sited at Congress Headquarters in the Great Hall of Red Cross House. The display will be of particular interest to members this year in view of the many recent therapeutic advances.

THE ARTS EXHIBITION

The Arts, Crafts and Hobbies Exhibition, which on this occasion will include the work of medical practitioners and their families, will be held during Congress week in the Art Galleries at the City Hall, Durban. Intending exhibitors should write immediately to the Convener of the Arts, Crafts and Hobbies Exhibition, Dr. M. J. Cohen, 82 Medical Centre, Field Street, Durban.

DISTINGUISHED GUESTS

The following visitors from overseas have accepted the invitation of the Organizing Committee to take part in the Scientific proceedings of Congress:

Sir Russell Brain, Bt., President, Royal College of Physicians of London.

Prof. T. Pomfret Kilner, C.B.E., Nuffield Department of Plastic Surgery, Oxford.

Prof. Alan Moncrieff, C.B.E., of the Institute of Child Health of the University of London.

Mr. A. J. Wrigley, Obstetric Surgeon to St. Thomas' Hospital and Medical School, London.

Prof. V. Kinross-Wright, Department of Psychiatry, Baylor University, Medical School, Houston, Texas.

SCIENTIFIC PROGRAMME

In preparing the Scientific programme, the Organizing Committee has encouraged the Sectional Secretaries to arrange as many combined sessions as possible while leaving sufficient time for individual Sectional Meetings.

The Business Meetings of the various National Groups of the Association will be held on Wednesday afternoon, 18 September, but should the Groups require more time, the last Sectional Meeting period on Friday afternoon could be used for adjourned Group Business Meetings. This would still leave time for three Scientific Sectional periods during the Congress.

The subjects of the two Plenary Sessions are *The Parasitic Diseases of Man in Africa* and *The Surgery of Repair*. Other symposia at Combined Meetings will include *Cerebral Vascular Disease* and *The Problem of Aging*, to be opened by Sir Russell Brain, of London, *Chemotherapy in the Treatment of Nervous and Mental Disorders* by Prof. V. Kinross-Wright, of Baylor University, Texas, USA, and *A Critical Review of the Report of a Thousand Maternal Deaths published by the Ministry of Health of England and Wales*, which is being presented by Mr. A. J. Wrigley, the Obstetric Surgeon to St. Thomas' Hospital, London.

Members wishing to read papers are reminded that a typed synopsis of the paper, not exceeding 500 words, should be in the hands of the Hon. Medical Secretaries not later than 31 May 1957, and a typed copy, in duplicate, of the complete paper not later than 30 June 1957.

ENTERTAINMENT

The Entertainment Committee, under the leadership of Mr. R. C. J. Hill and strongly supported by an active and enterprising Ladies Committee under the Chairmanship of Mrs. H. Grant Whyte, are doing everything in their power to provide a varied social programme and to ensure that everyone will be able to relax and enjoy their stay in Durban.

On Tuesday evening, 17 September, a Banquet for Medical Graduates will be held in the City Hall, Durban, at which His Excellency The Governor-General has been pleased to signify his intention to be present. This function will give members of the Association an opportunity of hearing and meeting informally our distinguished overseas visitors. Special arrangements will be made to ensure that fellow alumni, members of Groups, groups and friends, may sit together and have the opportunity of renewing acquaintainships.

On Thursday afternoon, 19 September, no Scientific Meeting will be held, and the Campbell-Watt Golf Trophy and the Bowls Challenge Cup will be competed for at Mount Edgecombe. There will also be facilities for tennis, sailing and fishing.

The Chairman of the Clairwood Turf Club has kindly offered Honorary Membership of the Club to members of the Association for their meetings to be held on Saturday, 14 September and Saturday, 21 September. The distinguished visitors will be the guests of the stewards.

The Durban Club has extended Honorary Membership to gentlemen of the Association and the facilities of the Ladies Dining Room. The Jewish Club has offered Honorary Membership to Members of the Association and their ladies. Facilities have also been offered by the Durban Country Club, the Royal Natal Yacht Club and the Kloof Country Club.

Intending visitors should book accommodation if they have not already done so. Accommodation is still available in Durban.

MORE SOUTH AFRICAN POLIOMYELITIS VACCINE ISSUED

BY OUR PARLIAMENTARY CORRESPONDENT

South Africa was again in a position to issue 80,000 doses of its own anti-poliomyelitis vaccine as from last week, according to an announcement made by the Minister of Health, Mr. J. H. Viljoen in the House of Assembly on Friday, 8 March 1957.

The Minister stated that of the poliomyelitis cases reported during the 6 months ended 28 February, 31 had had one injection and one had had 2 injections of anti-poliomyelitis vaccine.

The last supply of vaccine was made available for distribution on 21 December 1956. The Minister said, his attention had been drawn to a press report stating that supplies of Salk poliomyelitis vaccine from the United States could not be expected in the Union before 27 March and that the delay was due to the time taken by the United States Government to issue export licences. He had made representations to the United States Government through its Ambassador in the Union to expedite the granting of export permits.

Furthermore as the United States Government was not prepared to supply vaccine on an inter-governmental basis, his department had requested the United States Embassy in the Union to convey to its Government information required for the issue of export licences to commercial firms. In addition to these steps the Union's Ambassador in Washington was requested by cable on 20 February to convey to the United States authorities the information required in connection with the issue of export licences.

As all the information required by the United States authorities for the issue of export licences had been furnished by the Union's Department of Health, no further steps were contemplated at present.

Mr. Viljoen said he had given the United States Government the necessary assurances in regard to the distribution of the vaccine. He was unable to state what was the estimated date on which the vaccine would arrive in the Union as this was dependent upon the issue of an export licence by the United States authorities to the successful tenderer. No safety test would be made on the arrival of the vaccine but the supplier would be required to submit the protocols in respect of each batch of imported vaccine to his department for examination.

Mr. Viljoen said that during the 6 months ended 31 December 1956 there were 43 European and 23 non-European fatal cases of poliomyelitis. No statistics were at present available as to the number of cases during this period where the disease had caused paralysis. Mr. Viljoen said that it might be mentioned that of the 1392 cases notified during this period, the majority showed paralysis or paresis to some degree, in most cases slight. On the other hand by far the greater number of cases did not show the symptoms of paralysis and, therefore, escaped diagnosis.

QUESTIONS ANSWERED

Question: What are the effects of smoking on the cardiovascular system?

Answer: The Heart.

The effect of nicotine on the circulation is to increase the heart rate and the cardiac output; it may produce dizziness or faintness. Cigarette smoking appears to be more harmful than cigar or pipe smoking. There is no clear evidence, however, that smoking is harmful so far as the heart is concerned. Most patients find that smoking has no relationship to the production of their symptoms, particularly as regards angina pectoris.

However, in approximately one tenth of the patients angina may actually be induced by smoking. It is, therefore, wise to suggest to patients with angina pectoris that they should reduce or stop cigarette smoking temporarily, to see what effect this has on their condition. The attacks of pain may be more easily con-

trolled when smoking is eliminated. Should the patient definitely receive relief, he should be advised to stop smoking altogether, but it must be remembered that many patients find that when they stop smoking they gain considerably in weight. This in itself is disadvantageous; and, moreover, the loss of the comfort derived from smoking may also be disadvantageous.

In patients with pulmonary heart disease, smoking should be stopped because of the added effects of the irritant gases on the underlying lung disease.

Answer: The Peripheral Circulation.

There is as yet no unanimity of opinion about the possible injurious effects of tobacco on the vascular system and the part it plays in the etiology of certain peripheral vascular diseases. However, everyone who has had to deal with vascular diseases has been impressed by the following facts:

1. The rarity of cases of thrombo-angiitis obliterans amongst non-smokers.

2. Patients who continue smoking while being treated are extremely difficult to improve.

3. Patients who improve and refrain from smoking usually remain in better health than those who resume smoking. The latter are often soon back with new symptoms.

Smoking can exert its effect on the peripheral vessels in various ways, viz., by the direct effect of combustion products that are absorbed, and by indirect effects upon the vascular system *via* the vasomotor centre, and *via* the endocrine glands (adrenal). The act of smoking itself produces reflex vasoconstriction; this sometimes occurs on a single deep inhalation of smoke. A decrease may occur in the blood-flow of an extremity when a denicotinized cigarette is smoked or before there can be any absorption of nicotine when the usual type of cigarette is smoked.

In the sympathectomized limb, vasoconstriction from such reflex mechanisms cannot occur, and the reduction in the peripheral blood-flow in a sympathectomized limb which smoking produces is attributed to the direct pharmacological action of the absorbed nicotine or some other combustion product.

Some authors hold that it is an allergic reaction to nicotine which causes vascular disease.

Although there is no doubt that nicotine produces a reduction in the peripheral blood-flow, the role of smoking as an etiological agent in peripheral vascular disease is doubtful. It has not been proved that it can cause vascular disease, but there is little doubt that smoking may aggravate the condition in a patient who is already suffering from peripheral vascular disease.

As one of the aims in the treatment of vascular diseases must be to exclude all agents likely to cause a decrease in blood-flow, it is wise, with our present knowledge, to prohibit the use of nicotine in those who have contracted arterial disease.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Cyril Adler whose valedictory address as President for 1956 of the Southern Transvaal Branch of the Medical Association of South Africa appeared in the *Journal* of 9 March 1957 (p. 230), said he had adopted for his address a subject of deep concern to all members, that of the Medical Association in relation to its own functions, and those of the South African Medical and Dental Council in relation to the Medical Profession. He had chosen '*Quo Vadis*'? as the title because he was not quite sure where they were going and—what was perhaps more important—what was the ultimate destination of the Medical Profession and the results of the activities of the Medical Association. Dr. Adler spoke of the economic aspect of the practice of medicine and the growing tendency to lay control of medical practice. The public demanded the best medical services and it would only be able to obtain such services with the help of and through the Medical Association of South Africa.

Dr. Cyril Adler

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Dr. C. K. Schwarz, who was recently admitted as a Member of the Royal College of Physicians in London, has been appointed lecturer in Public Health at the Liverpool University School of Hygiene and Honorary Assistant Medical Officer of Health for the City of Liverpool.

* * *

Dr. Jacob Adno, M.B., B.Ch., Dip. O. & G. (Rand) has commenced practice as an obstetrician and gynaecologist at 704 Medical Centre, Jeppe Street, Johannesburg. Telephones: (the numbers are not yet in the telephone directory), rooms 28-8491, residence 45-5436.

Dr. Gerald Kane, M.B., B.Ch. (Rand), F.R.C.S. (Eng.) Spesialis-Chirurg, praktiseer nou by die volgende adres: Sanlam-Gebou 415-416, Smithstraat, Durban. Telefone: Spreekkamers 2-4041, Woning 4-1615, Na ure 2-9326.

Mr. Gerald Kane, M.B., B.Ch. (Rand), F.R.C.S.(Eng.) is now in practice as a Specialist Surgeon at 415-416 Sanlam Building, Smith Street, Durban. Telephones: Rooms 2-4041, Residence, 4-1615, Emergencies 2-9326.

* * *

Dr. Neville Sacks, B.Sc., M.B., Ch.B. (Aberd.) who was recently admitted a Member of the Royal College of Physicians of Edinburgh, is the son of Dr. and Mrs. I. Sacks of Bloemfontein. Dr. Neville Sacks at present holds an appointment at Grey's Hospital, Pietermaritzburg.

* * *

Dr. Adolph Meyer, Radiologist (Drs. Meyer, Latham and Tucker) wish to draw attention to an error in the new telephone directory. His address remains 702 Netherlands Bank Buildings, 85 St. George's Street, Cape Town, and *not* Medical Centre, as stated in the telephone directory. The telephones, which remain unchanged, are 2-1290 and 3-5464.

* * *

Mr. I. Norwich, Part-time Head of Department of Surgery, Edenvale Hospital, Johannesburg, is proceeding overseas on 12 March 1957 for 3½ months. He has been invited to observe the Surgical Clinic in New Orleans and to work in the Surgical Department of Mount Sinai Hospital, New York. He will also visit hospitals in London. Mr. Norwich will be accompanied by his wife.

* * *

Health Education. The Central Council for Health Education is holding 2 residential seminars in England and Wales during 1957 for public health workers from overseas.

Medical Director, Dr. John Burton, Tavistock House North, Tavistock Square, London, W.C. 1, to whom all communication should be addressed.

The first seminar will be held on 15-19 April 1957 at Passfield Hall, Endsleigh Place, London, W.C. 1. It is arranged primarily for delegates attending the annual Congress of the Royal Society of Health, and is designed to provide workers from overseas with the opportunity of studying the broad principles of pro-

gramme planning, evaluation and organization, and the use of methods and media. The titles of subjects that will be discussed include Fundamentals in health education, The unity of content and method, Community attitudes and motivations, Selection and use of materials, Mass media in health education, Posters and leaflets.

Delegates will be accommodated at Passfield Hall in single bed-sitting rooms with hot and cold running water in each room. The fee for tuition and residence, inclusive of gratuities, is £8 8s. 0d.

A summer school in health education will also be held on 20-30 August 1957 at Neuadd Reichel (Reichel Hall), University College of North Wales, Bangor. This is a working conference for doctors, nurses, health inspectors, teachers, and auxiliary health and welfare workers from various countries. It will have similar aims to the April seminar, and will offer an intensive practical course. Students will be housed mainly in individual study bedrooms with hot and cold water in each room. The inclusive fee will be £21 0s. 0d. (tuition fee £9 14s. 6d., residence £11 5s. 6d.). Deposit of £2 2s. 0d. of the total fee is payable on registration and is not returnable after 20 July except in cases of personal illness, when a medical certificate will be required.

* * *

South African Medical Congress, Durban, 16-21 *September* 1957. *Scientific Exhibition Section.* Mr. B. W. Franklin Bishop, convener of the Scientific Exhibition, invites anyone who is interested in assisting with an historical exhibit in connection with the development and growth of medicine in the Union of South Africa, documents, instruments, photographs, etc., to communicate with him at 112 Medical Centre, Field Street, Durban.

Bowls Tournament. Mr. B. W. Franklin Bishop, convener of the bowls tournament, invites bowlers who will be attending the Congress to send their names to him at the above address as participants in this tournament, for which there is a trophy.

* * *

Union Department of Health Bulletin. Report for the 7 days ended 28 February 1957.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Bombay, Calcutta (India).

Smallpox in Rangoon (Burma); Ahmedabad, Alleppey, Bombay, Calcutta, Cuddalore, Delhi, Jodhpur, Kandla, Kanpur, Nagapattinam, Quilon, Tiruchirappalli, Visakhapatnam (India); Baghdad, Mosul (Iraq); Chalna, Dacca, Karachi (Pakistan); Nairobi (Kenya).

Typhus Fever in Baghdad (Iraq).

* * *

Aangesien dr. Alice Cox uit private praktyk getree het, praktiseer dr. Benjamin Chesler vanaf Desember 1956 as psigiater saam met drs. Max Feldman, Carl Jeppe en Fred Frankel te Keyeshof 1, Keyeslaan, Rosebank, Johannesburg. Telefone 42-2481, 42-2153.

Dr. Chesler het ook die praktyk van wyle dr. Felix du Toit oorgeneem.

REVIEWS OF BOOKS : BOEKRESENSIES

CELLULAR PATHOLOGY

New Pathways in Cellular Pathology. By Gordon Roy Cameron, M.B., D.Sc. (Melb.), F.R.C.P., F.R.S. Pp. vii + 90. 42 Figures. 16s. net. London: Edward Arnold (Publishers) Ltd. 1956.

Contents: I. Introduction. II. The Cell Membrane and Surface. III. The Cytoplasm. IV. The Nucleus. V. Mitochondria. VI. Microsomes and other Cell Components. VII. A New Form of Pathology. (i) Ideas from Toxicology. VIII. A New Form of Pathology. (ii) Some general pathological processes reconsidered. Index.

As students all medical men become familiar with cloudy swelling and fatty degeneration and come to regard them as among the early visible pathological changes in a previously normal cell. They were changes visible under the microscope but, although accepted at face value and fitted into the pathological progression of event, they were never convincingly explained. No one really knew how these visible degenerations were brought about.

In this stimulating small book Professor Cameron has partially lifted a blind and let some light into a dark room. He outlines the research which is being pursued at the present time and provides some details of methods employed. He is able to indicate some of the biological functions of the cell components, placing them in positions of relative importance. He is also able to show the sites at which present opinion places some of the common enzymic reactions. Moreover, their probable position in chains of events leading to morbid histology is explained. He quotes (among others) two important pieces of research which were directed by Dr. Peters and which have come into the orbit of most medical men, namely the discovery of British anti-lewisite and the mechanism of lethal synthesis which so to speak 'hoodwinks' nature and renders the South African plant *gifblaar* toxic.

This is an unusual book, which holds one's attention and hints strongly at future research into some fundamentals of a new approach to pathology.

H.W.C.

SEX

The New Commonsense About Sex. By Leonora Eyles. Pp. 96 + 3 figures. 6s net. London: Victor Gollancz Ltd. 1956.

Contents: 1. Finding Out. 2. Are You Courting? 3. Getting Married. 4. Sexual Intercourse. 5. Difficulties in Marriage. 6. Infidelity. 7. Birth Control. 8. Masturbation. 9. Some Problems.

This is a straightforward little book which does not go into any great detail but discusses its subject in a very frank and friendly manner. The chapters on anatomy and physiology are skimped but the book does not claim to be a complete one and is indeed rather an introduction. It can be thoroughly recommended to any doctor to give to such of his patients as come for advice on this matter.

T.S.

CORRESPONDENCE : BRIEWERUBRIEK

THE CIVIL CASE AGAINST DR. M. GODDEFROY

To the Editor: For many years the practitioners of the Free State were twitted about the droughts, pestilences, freezing nights and scorching days of its plains, till the potential wealth of its gold mines roused more favourable comments from the colleagues in the other provinces of the country. Even we, of the Cinderella province, believed that those qualities of neighbourliness and kindness that characterized the citizens of our veld would be submerged under the impact of industrialization after the discovery of its gold. But the recent case against Dr. Goddefroy proved the contrary, and the staunch support given by medical colleagues to one of their number in distress deserves the widest publicity.

Dr. Marius Goddefroy is a typical example of the best type of general practitioner—courteous, quiet, unassuming, charitable, with an honourable sense of duty and consideration for his patients. Our whole medical community was shocked one morning recently at the news in the daily press of this litigation against our respected colleague. A few of Dr. Goddefroy's intimates may have been aware of this sword of Damocles which was hanging over his head, but the rest of us were shocked to read that he faced a claim for £20,000 because of alleged negligence in prescribing 'cafergot', which was said to have damaged the plaintiff's eyesight. The

outcome of the case, an unconditional withdrawal by the claimant of her allegations of negligence, with an apology for any inconvenience that may have been caused to Dr. Goddefroy, was amply recorded in the press. But there is another aspect of the case which did not receive sufficient publicity, and deserves to be stressed.

Though the report in the press stated Dr. Goddefroy won his case with costs, it was never mentioned that as a result of his response to an approach made by the claimant concerning her financial position, Dr. Goddefroy found himself with £3,600 to pay in costs. His insurance policy gave him cover for only £1,000, which meant he had to find the balance of £2,600.

The legal implications of this case to our profession will, I trust, be discussed by our Federal Council, and I will confine myself to the reaction of the medical men of Bloemfontein. Each of us here felt that he might be involved in a similar misfortune, and that the case affected not only Dr. Goddefroy, but the whole body of medical men in this country.

No meeting was held to discuss the various aspects of the case, but a list appeared asking for contributions. It exceeded the hopes of the promoters to find that the medical men of Bloemfontein had contributed over £1,000. This magnificent gesture deserves the widest publicity in an age where self-interest and materialism have to a great extent ousted charitable feelings towards our fellowman.

This heart-warming response to a fellow colleague's distress reflects the tenets of our South African motto *ex unitate vires*, and it is in the hope that other colleagues throughout the country will feel they would like to contribute towards the final liquidation of our colleague's legal obligations that this letter is submitted to the *Journal*.

C. D. Brink

63 South African Mutual Building
Bloemfontein
21 February 1957

THE CAPE TOWN CITY COUNCIL ADVERTISEMENT

To the Editor: An advertisement appeared in the *Journal* on 9 February 1957 in which the City Council of Cape Town call for services of 15 European general practitioners to attend to employees contracting illnesses or sustaining injuries during and in the course of their employment with the City Council.

The City Council has attacked two fundamental principles in this advertisement, namely:

(*a*) The right of a duly registered medical practitioner, irrespective of his supposed racial origin, to attend to any person who has sought his services.

(*b*) The patient's right of freedom to choose his own doctor.

The Council has implied that it is undesirable for doctors not classified as Europeans to attend to its workmen. Why? Inferior qualifications? They have all either qualified at the two leading South African Universities or at leading Universities overseas. Inherent inferior ability? Perhaps even the gentlemen concerned in the City Council will shy from such assertions (at any rate the professional racialists are beginning to). Or will they claim to be bowing to the 'known prejudices of White South Africans'?

Nevertheless, whatever their reason for their preference for European practitioners we feel that it is the duty of the Association and the *Journal* to come out openly in defence of members of the medical profession when they are being discriminated against on racial grounds. (This is but one of many instances).

Similarly the Association should on this occasion have denounced the closed-panel system in general, with special reference to the attempt of the City Council to create a closed panel for treating cases falling under the Workmen's Compensation Act. This is contrary to the spirit of the Act; and it is furthermore a serious inroad into one of the last fields where the worker has a free choice of doctor.

The Association has on previous occasions taken a firm stand on the question of the closed-panel system and the fundamental right of the patient's free choice of doctor. But why encourage exactly the opposite view by publishing an advertisement to abolish this right for thousands of workers? The least we could have expected was a statement of policy on this question in the subsequent *Journal*, and not merely a request for intending applicants to get in touch with the Secretary so that they could be informed privately of the Association's attitude and its behind-the-scenes manoeuvres.

D. Clark, M.B., Ch.B. (Cape Town)
I. D. K. Davids, B.Sc., M.B., Ch.B. (Cape Town)
M. A. Ebrahim, M.R.C.S. (Eng.), L.R.C.P. (Lond.)
41 Frere Street — P. M. Heneke, M.B., Ch.B. (Aberd.)
Woodstock — V. K. Moodaley, L.R.C.P. & S. (Edin.), L.R.F.P.S. (Glasg.)
24 February 1957

PRESIDENT OF CAPE WESTERN BRANCH EXPLAINS

To the Editor: On 9 February 1957 an advertisement inserted by the Cape Town Municipality appeared in the *Journal*. This advertisement asked for applications for 15 European medical officers to act on a panel to attend municipal employees who were injured or became ill while on duty. I have been asked as President of the Cape Western Branch to state the attitude of the Branch to this advertisement and to outline the action taken by the Branch Council.

It must be obvious from the outset that the suggested scheme violates the principle of the open panel, or free choice of doctor.

It is further noted that the discrimination, on racial grounds, implied in the advertisement, involves a limitation of opportunity to non-European doctors in a field where a substantial majority of the patients are non-European.

Finally there seems to be some doubt as to the propriety of accepting an appointment in which one authority (the Municipality) secures the patients for the doctor while another authority (the Workmen's Compensation Commission) pays the fees.

The first question posed by enquiries is, 'Why was the advertisement published?' The answer to this is that while it is possible to delay the insertion of an advertisement for discussion with the advertiser and with the appropriate Standing Committee of Branch Council, all advertisements of appointments submitted for publication must actually be published, whether they secure approval or not. Municipal, Government and other official advertisements are usually accepted as correct. They are outside the ambit of ordinary contract practice. The normal routine is to refer doubtful advertisements to the Branch Council for further consideration. In this case it was the appearance of the advertisement which first drew the Branch Council's attention to the proposed appointments.

The next question is: 'The notice having appeared, what did the Branch Council do about it?' As soon as attention was drawn to this advertisement, the Editor was asked to insert an important notice in the *Journal*, requesting members to communicate with the Honorary Secretary before accepting one of these appointments. This notice appeared on 16 February.

The matter was discussed at meetings of the Executive Committee of Branch Council on 12 February, and of Branch Council on 15 February. The Honorary Secretary was instructed to inform enquirers that they should not apply for or accept the appointments, and a deputation consisting of Dr. Bailey, Dr. Zabow and myself was instructed to interview the responsible Committee of the City Council and to secure withdrawal of the proposed scheme. The deputation met the Deputy Town Clerk, stated their objections to the scheme, were courteously received, and were asked to interview the Committee responsible for the advertisement. The deputation is to meet this Committee on 13 March.

In the meantime a special general meeting of the Branch was held on 8 March. After full discussion, this meeting resolved: (1) To express its unequivocal opposition to the appointments. (2) To ask for more vigilant scrutiny of such advertisements in future. (3) To send a deputation to the City Council to secure withdrawal of the advertisement and to see that this withdrawal was published in the *Journal*. (4) To emphasize the opposition of the Branch to the racial discrimination implied in the advertisement. These resolutions are briefly summarized here and actually cover a wider field than can be indicated in this letter. The resolutions and arguments put forward at this meeting will form the basis of representations to be made by the deputation on 13 March.

J. A. Currie
President
Cape Town — *Cape Western Branch*
11 February 1957